Clinical Aspects of the Electrocardiogram

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St. Joseph's Hospital.
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CLINICAL ASPECTS OF THE ELECTROCARDIOGRAM

Clinical Aspects of the
ELECTROCARDIOGRAM

A MANUAL
FOR PHYSICIANS AND STUDENTS

BY

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WITH SIXTY ILLUSTRATIONS

SECOND EDITION
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To
PROFESSOR HORATIO B. WILLIAMS
in grateful recognition of
his contributions to the
electrocardiographic
method

PREFACE TO SECOND EDITION

Since the first edition of this book no new basic principles in the clinical use of the electrocardiogram have appeared. It has become increasingly evident, however, that this method of examination of the heart is of great importance. It has been found to give evidence of involvement of the myocardium in numerous disease processes. The acute degenerative changes that follow thrombosis of a branch of a coronary artery, the chronic degenerative changes that result from the gradual narrowing of a single branch or from generalized coronary arteriosclerosis, the acute toxic myocardial degeneration due to diphtheria and the acute inflammatory changes associated with rheumatic fever; all these disease processes, and other less common ones as well, give signs in the electrocardiogram. At times these will be the only discoverable signs of heart disease.

Additions and changes will be found in the text so that this volume presents the important acquisitions of knowledge during the last three or four years. Possibly the most striking change is the adoption of a new terminology to express the variations of the Q R S group which Einthoven attributed to hypertrophy of the right or the left ventricle. It is believed that when the idea of the electrical axis of the Q R S group is thoroughly grasped the advantages of this conception will be found to be compelling.

The mechanical developments of the last few years have given us several new models of the string galvanometer, and satisfactory portable instruments are now available. A new principle of recording the electricity of the heart beat has come into use. The familiar radio amplifier tube is used to increase the power of the heart's current so that a less fragile instrument than the string galvanometer may be used to record the electrocardiogram. These mechanical features are given brief but sufficiently explanatory mention.

New York, N. Y. HAROLD E. B. PARDEE.
February, 1928.

PREFACE TO FIRST EDITION

The use of electrocardiographic records in the diagnosis of heart disease has been but slowly developed. It was necessary first to determine what the waves of a normal record represented and then to observe a large number of abnormal records in connection with the clinical features of the patients who gave them. Our present knowledge of the interpretation and prognostic importance of the electrocardiogram rests upon such a large number of observations by so many different authors that the beginner is unable to acquire it without consulting a widespread and voluminous literature. I have therefore thought it worth while to collect together in this volume all the current knowledge of the electrocardiogram which is of clinical importance, leaving the details of experimental and theoretical considerations to the special workers in this field. Yet experimental observations and theories cannot be entirely ignored by the clinician, for they give a perspective which clarifies many of his clinical observations. They are therefore included here in as simple a form as possible.

The limits of the normal variations of the electrocardiogram have been carefully described, for only by being familiar with the great variety of these can one distinguish certainly the abnormal curves. The clinical aspect of these abnormal curves has been kept constantly to the fore, that the physician may know what sort of information he may expect to obtain from the records. The electrocardiogram affords but one of many methods of examining the heart and though it gives information which cannot be obtained in any other way regarding the contraction of the auricles and ventricles, yet in order to get the full value from the records, they must be correlated with the complete history and physical examination of the patient. I have tried to point out the detail of this correlation. Abnormal electrocardiograms are considered as an expression of abnormal muscle function, and the possibility of an underlying disease is then discussed.

The basic electrical and mathematical features of the method have been explained by diagrammatic drawings which may help the reader to obtain the necessary conception of the electric activity of the heart. This technical discussion has been kept almost entirely within the bounds of Chapter IX. Perhaps the reader who finds in it an unfamiliar vocabulary will be better prepared to read this chapter after having familiarized himself with the clinical aspects of the records presented in the earlier chapters. He will probably find it to be more easily comprehended than he had thought.

Controversies have not been discussed in detail. The opinion which seems most sound has been presented, with a mere statement of the opposing views. The references for supplementary reading are intended for those who wish for more detail. They do not form a complete bibliography of the subject—this would be too cumbersome to be useful—but give the important standard sources and the best of the newer work.

So little has appeared in the literature about the technique of using the electrocardiograph that it was thought worth while to add a chapter on the construction and operation of the instrument. Those features which might lead to the taking of incorrect records are especially emphasized and it is pointed out how they may be avoided.

It is a pleasure to acknowledge my indebtedness to Professor Horatio B. Williams of the Department of Physiology of the College of Physicians and Surgeons, Columbia University, for his invaluable instruction during my years as his assistant. Without the grounding in the fundamentals of electrocardiography received from him, the clinical observations of later years would not have been possible.

I wish also to acknowledge the kindness of Dr. Lewis A. Conner and Dr. W. R. Williams for the privileges afforded in the study of clinical material in the New York Hospital, where most of the records used to illustrate this book were obtained. I feel that a final word of thanks is due to Mr. Paul B. Hoeber and his editorial staff for their interest in the manuscript of this book and for their many helpful suggestions in the matter of arrangement.

HAROLD E. B. PARDEE.

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Chapter I

INTRODUCTORY

The clinical study of the electrical current accompanying the heart's contraction was made possible by Einthoven of Leyden, in 1904, by the perfection of an instrument sufficiently sensitive and quick to follow the small, rapidly varying currents produced by the heart. It had been known since 1856 that the contraction of the heart was accompanied by the production of an electrical current. In 1887 Waller demonstrated that this current could be led off from the surface of the body and recorded, if only a proper contact were made between the wires from the galvanometer and any two areas of the body including the heart between them. At that time, however, the medical world was not prepared to consider seriously so complicated a diagnostic appliance. Even the physiologist had difficulty in making use of this knowledge, for the only instrument available was the capillary electrometer, which was totally inadequate to record with any degree of accuracy the quickly varying currents of the heart beat. Einthoven's string galvanometer was first described in 1903 and in 1906 and 1908 he published the results of his first clinical studies. The clinical use of the electrocardiograph soon became general in the large clinics abroad and the first instruments were installed in this country between 1910 and 1913.

The record of the string galvanometer consists of a series of deflections or waves (Fig. 2) produced photographically by the up-and-down movement of the shadow of the string of the instrument. When the patient is connected with the instrument, the shadow of the string can be seen to jump up and down in response to the current from the heart, which is passing through the patient's body, the wires and the string itself. These movements are photographed by the simple process of drawing along behind a slit through which the light from the galvanometer passes and across which falls the shadow of the string, a strip of film, or bromide paper. The continual movement of the photographic film behind the slit converts the up-and-down jumps of the string shadow, which we can see, into the up-and-down waves of the record. An upward wave indicates a current in one direction, a downward wave a current in another. A straight line indicates that no current is passing, for the string shadow then remains still as the photographic film moves behind the slit. Vertical lines across the record will indicate time, because as the paper moves onward successive vertical sections of it lie behind the slit.

The Three Leads

The fundamental fact, as Waller demonstrated, is that if two wires are connected with two areas of the skin through some appropriate medium, such as cloth pads wet with salt solution, and these wires are connected to the galvanometer, the instrument will record a current if the two skin areas are *both* very near the heart. If some distance from it, these skin areas must include the heart between them. Any pair of areas from which the current is led off to the instrument is called a *lead* or *derivation*. Almost every different lead carried a different sort of electrical current from the same heart. Some leads were favorably situated to record the heart's current; some leads were unfavorably situated. It therefore became necessary to adopt standard leads in order to facilitate the comparison of records from different patients. Leads from the extremities have been universally used for clinical work and these were selected because they were relatively favorable leads and because of the ease of applying electrodes to the extremities. Only three of the six possible combinations of the extremities are used, as follows:

Right arm—left arm called Lead 1
Right arm—left leg called Lead 2
Left arm—left leg called Lead 3

Lead 1 is horizontal in relation to the body, Lead 2 is oblique and Lead 3 is in the left lateral position and is more or less vertical. This will be clear if we consider the relation of the two shoulders and the left hip to each other; and it is really from the shoulders and hip that the current is led off from the trunk itself (Fig. 1).

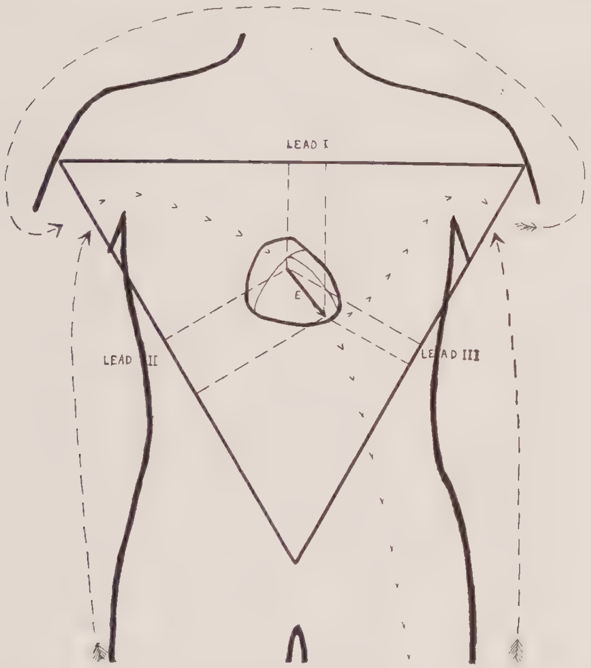

FIG. 1. Illustrating the relation of the current within the heart to that obtained by the three leads. The body is viewed from the anterior aspect. Each side of the triangle between the right arm, the left arm and the region of the legs (left leg) represents one lead.

A current within the heart (arrow E) would be represented in each lead by excursions of the same size relative to each other as the projections of this arrow upon the sides of the triangle. The line of arrowheads passing through the body from right arm to left arm represents the flow of current in Lead I(1), and the circuit is completed outside the body through the galvanometer as indicated by the long dotted arrow between the two arms. The flow within the body is indicated for Lead II(2) by a line of arrowheads passing from right arm to left leg. The return flow through the galvanometer for Lead II(2) is shown by the dotted arrow between the leg and right arm. The flow through the galvanometer for Lead III(3) is indicated by the arrow between the leg and left arm.

In each lead the extremity nearer to the apex of the heart is called *apical*, while the other, nearer to the basal region, is called *basal.* The wires from the extremities are connected to the galvanometer so that a current passing through the galvanometer from the apical to the basal extremity of each lead will produce an upward movement of the line of the record. (Dotted arrows of Fig. 1.) This standard method of connecting the patient with the galvanometer terminals insures the waves having the same direction in each lead each time the record is taken. If a wave should be found downward instead of upward it is because of a change in direction of the electricity of the heart and not because of a change in the attachment of the patient to the instrument.

The Size of the Deflection

In order to be able to compare different records it is not only necessary to use the three standard leads, but also to standardize the galvanometer *before taking each lead,* so that the same amount of excursion will always result from the same strength of current passing from the patient. The technique of standardization is described in detail in Chapter X. It will suffice the clinician to know that in every properly taken record a movement of the string shadow of 1 cm. above or below the base line means that there is 1 millivolt of electricity in the galvanometer circuit.[1] Amounts of other strength are represented by deflections *proportionately* larger or smaller. The deflections may be small in any one lead if that lead happens to be unfavorably situated for recording the heart's current. The other leads will show larger deflections in such a case, for only one of the three standard leads can be unfavorable in a given patient (see Lead 3 of Records G and H of Fig. 5). Records should include a control curve of the standardization, for by an inspection of this the exactness of the instrument and of the technique of the operator can be determined (Chap. X).

The Nomenclature of the Waves

Figure 2 is a record taken with the string galvanometer by the three leads. The leads are not taken simultaneously, as might appear from the illustration, but one after another, the whole process consuming from three to five minutes. The series of steps at the left end of each lead is the control of

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[1] Upward deflections are measured from top of base line to top of wave; downward deflections from bottom of base line to bottom of wave.

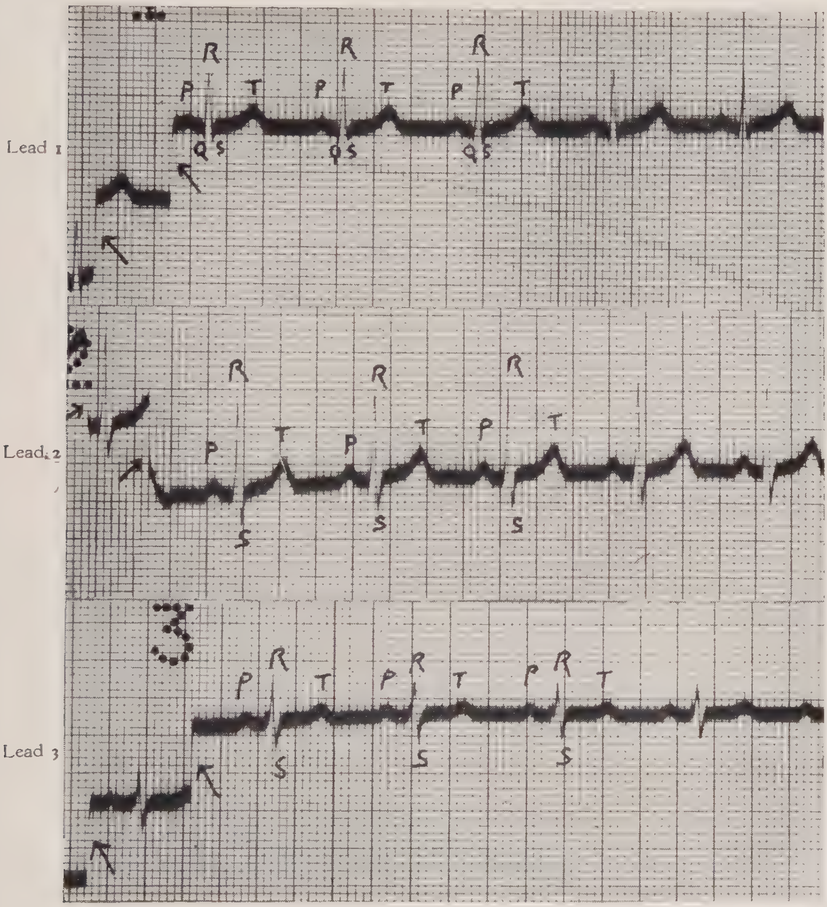

FIG. 2. Electrocardiogram of a normal individual taken by the usual three leads.

In all records, the horizontal lines represent strength of current: one division = 0.1 millivolt (.0001 volt). The vertical lines represent time units, and when forming a system of small squares, as in this record, each line represents .04 sec.

The series of steps indicated by arrows at the left end of each lead is a test of the standardization of the instrument, performed by the operator as described in the text (p. 201).

the standardization. Each jump of 10 mm. is due to a difference of 1 millivolt in potential applied by the operator.

Throughout each lead is a level which might be called the base line or zero level, and there are groups of waves (deflections) pointing upward and downward from this base line. Each group of waves represents one complete cycle of contraction of the heart and is called an electrocardiogram. The letter opposite the peak of each of these waves has been adopted as the conventional name for the wave.

Each heart cycle is ushered in by a small rounded elevation called P. This is followed by a series of sharp upward and downward deflections which are called the Q R S group. If there is an upward deflection it is called R; if there is a downward deflection it is called Q when it *precedes* R, and S when it *follows* R or when no R is present. Following the Q R S group there is a short interval during which the line of the record rests close to zero. It then curves upward or downward to form the final deflection called T. A broad, low wave called U is sometimes observed following T. It is probably not due to the heart at all, but is caused by the great vessels. If U is not present the line of the record remains at rest until the next P.

Though P and T are usually upward deflections as just described, this is not always so. Either or both may be directed downward in one or more of the leads or may be so small as to be almost indiscernible in one lead. Such variations are due to the relation of the lead to the current in the heart. If the wave is turned downward it is spoken of as an inverted P or T wave. If the Q R S group shows its chief deflection upward, the peak is called R, as has been said. The chief deflection of Q R S may, however, be downward in one or more leads. If this deflection precedes R it is called Q, while if it follows R it is called S. It is *not* customary to speak of an inverted or downward R wave.

Deflections Not Due to the Heart

Two rather common causes of distortion of the record must be mentioned in order to point out that they are independent of the action of the heart. One is the fine vibration of the line of the record which is seen in Figure 16 B and in Lead I of Figures 12 A and 15 B. This at times may be so marked as to obscure the waves of the electrocardiogram. It is due to the activity of the voluntary muscles of the extremities, though not necessarily to actual movements of the limb. If the arm or leg is held stiffly, as with a patient who is a little apprehensive, or if the body or the limb is cold and the shivering reflex evoked, these vibrations will be very noticeable. The other distortion, seen in Figure 29 B, consists of an upward or downward movement of the base line of the record. This slow wave-like movement is due to the reflex vasomotor effect upon the skin vessels of a varying mental state. The skin change causes a change in the electric potential of the limb in some way which is not understood, and this causes the wandering movement of the base line. With a nervous patient this movement is almost continuous and makes it very difficult to standardize the instrument so that the deflection from 1 millivolt will be exactly 1 cm.

Sources of Error in Technique

At the beginning of the record of each lead in Figure 2 is the control of standardization. The base line of the record is suddenly deflected upward or downward for ten scale divisions, which in the original record are each 1 mm. This is done by the operator who turns in or out a potential of 1 millivolt (0.001 volt) in the electric circuit containing the patient and the galvanometer. The resulting deflection should be exactly 1 cm., should be completed in .02 sec. or less and should not show any overshooting of the string before finding its new level. (See Chap. X for an explanation of this.) If these requirements are not complied with by the control, an error in technique is indicated, which will result in the record being a more or less inexact picture of the true electrocardiogram of the patient. It is a less serious error in technique if the deflection of the control should fail to be 1 cm. than if the deflection should take longer than .02 sec.

or if there should be overshooting. The first can be easily corrected by adding to or subtracting from the height of the wave in the record the percentage error in the control, e.g., if the control deflection is only 8 mm. instead of 10 mm., then each deflection in the record should be 25 per cent larger than it is. A slow deflection time or the presence of overshooting in the deflection will result in a distortion of the record for which it is very difficult to make proper correction.

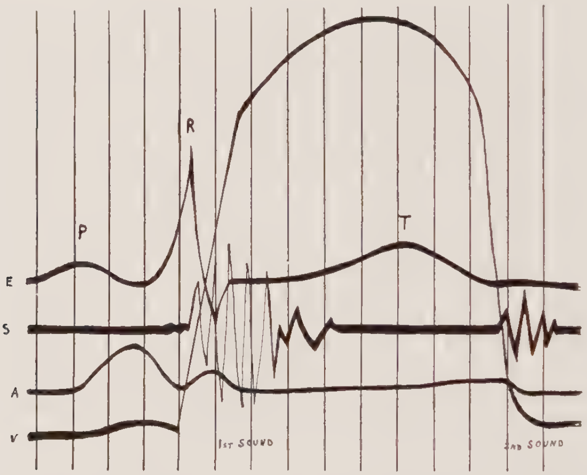

Fig. 3. Simultaneous records of the electrocardiogram (E), the heart sounds (S) and the pressure changes within the right auricle (A) and the left ventricle (V). These are represented within the same system of time lines as if obtained from the same heart beat, each line representing .04 sec.

Note that the P wave of the electrocardiogram begins before the rise of pressure within the auricle, and the Q R S group before the rise of pressure within the ventricle and slightly before the beginning of the first heart sound. The end of T slightly precedes the second heart sound and the sharp drop in intraventricular pressure.

Physiological Origin of the Waves

The series of waves and peaks of the electrocardiogram are due to the physiological activity of the heart-muscle fibers. It is not a special characteristic of the heart to produce electricity. The contraction of any muscle in the body will give rise to an electrical potential which the string galvanometer will record if electrodes are applied so as to lead the current to the instrument. The heart muscle, being of complex structure, produces a very complicated series of waves with each heartbeat. Each group of waves is an electrocardiogram. It is spread out so that the systole, which takes about .50 sec., occupies 12 or 13 mm. of the record, while the diastole occupies 5 mm., more or less, depending upon the heart rate. A record of these waves, obtained by each of the three standard leads, constitutes *an electrocardiographic record* as used in clinical medicine (Fig. 2).

Figure 3 has been constructed to give an idea of other activities of the heart at the time of each wave. It shows other events of the heart cycle in the same system of *time lines* as the electrocardiogram. The upper curve (E) is the electrocardiogram taken by Lead 2. Below this (S) is a record of the heart sounds as if taken simultaneously by means of a microphone and a second galvanometer. The third line (A) is a curve of the pressure variations of the blood within the right auricle and the fourth (V) shows the pressure variations of the blood within the left ventricle. The curves of this diagram were properly placed within the time lines by reference to such synchronously taken records as could be found in current physiological literature.

It is necessary to emphasize that the auricles and ventricles do not contract and relax as a single unit. First the two auricles contract simultaneously and then, after a slight pause, the two ventricles. The electrical effects of the auricular and ventricular contractions are quite separate from one another. Even within these units the fibers do not start their contraction all at once. Certain fibers receive the stimulus before others and these contract first and are probably the first to relax. This relatively gradual onset of the contraction of the muscle fibers in the wall of each chamber, will cause the rise of pressure within the chamber to lag slightly behind the first evidence *of muscle activity* which is the production of electricity. Accordingly the electrical wave due to the auricular contraction is seen to precede the rise of pressure within the cavity; and the same is true of the ventricles. The beginning of the fall in pressure does not mark the end of the muscular activity of the auricles or of the ventricles, but marks only a predominance of the relaxing fibers over those that have not yet relaxed. Accordingly the electrical curve continues after the pressure begins to fall.

The P wave of the electrocardiogram starts just before the rise of pressure within the auricle (Fig. 3) and, since the two auricles contract simultaneously, this wave must be referred to the beginning of the contraction of the muscle fibers of the walls of these chambers. The P wave is completed before the auricular contraction has reached its end, the end of P coinciding with the top of the rise of intra-auricular pressure.

The series of quick waves marked Q, R and S should be considered as a group rather than as individual waves, as was customary with most of the older writers. The reasons for this will appear more plainly later. This group of waves is seen to begin before the rise of intraventricular pressure and even before the first heart sound, and records the electrical changes with the beginning of the ventricular contraction.

Some writers have considered the P wave and the Q R S group as due to the process of "excitation" which theoretically precedes the process of contraction. There does not seem to me to be any advantage from such a conception of the cause of these electrical changes, especially as there is no evidence, experimental or otherwise, that "excitation" is sufficiently distinct from "contraction" to warrant our making such a separation of their electrical processes. As long as we realize that these waves record the electrical accompaniment of the earliest chemical changes of the contraction process, it makes little difference whether we speak of it as excitation or as contraction. I feel that the introduction here of the term "excitation" serves no useful purpose and may even mislead, constituting an undesirable complication of terminology.

The marked difference between the form of the P wave and that of the Q R S group depends upon the difference in the way in which the contraction stimulus is distributed to the auricles and the ventricles. The auricles contract in response to a stimulus which is formed in the sinoauricular or sinus node. This is about 3 cm. in length and lies in the wall of the right auricle just below and in front of the entrance of the superior vena cava (Fig. 4). The contraction spreads radially from this area throughout the auricular muscle, involving the two chambers about synchronously. Arriving at the base of the interauricular septum, the contraction produces an effect upon the auriculoventricular node, which starts an impulse, probably also a contraction, along the auriculoventricular bundle (bundle of His) toward the ventricles. This bundle divides upon the top of the interventricular septum and one branch passes down into either ventricle. The one to the right side runs in the wall of the septum toward the septal papillary muscle of this ventricle,

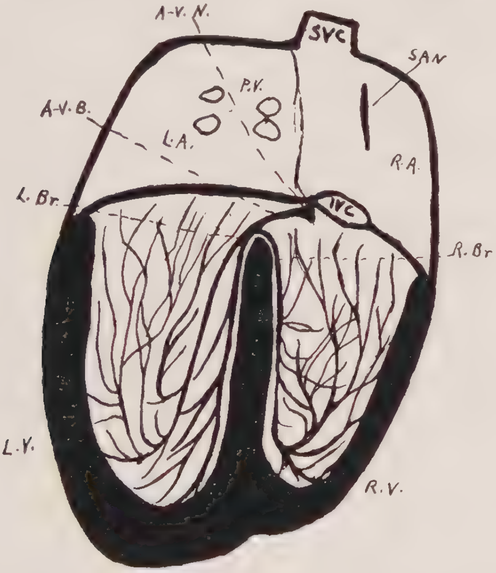

FIG. 4. A diagrammatic view of the heart from behind and somewhat below. The superior vena cava (S. V. C.) and the inferior vena cava (I. V. C.) are seen entering the right auricle. Mouths of the pulmonary veins (P. V.) are seen entering the left auricle. The sinoauricular node is indicated.
The ventricles are represented as if cut open from behind so as to show the auriculoventricular node (A. V. N.), and the auriculoventricular bundle (A. V. B.) of His, and its branchings in the right and left ventricles. L. Br. = left branch. R. Br. = right branch.

comes to the surface of the endocardium below this and quickly breaks up into a fine network spreading over the whole inner surface of the ventricle. A rather large branch passes to the anterior papillary muscle. The left branch of the bundle enters the left ventricle beneath the right cusp of the aortic valve, and lying superficially beneath the endocardium of the left side of the septum, quickly divides into two main branches. One of these passes toward the base of each of the papillary muscles of the left ventricle, sending off many branches on the way, which quickly break up into a network all over the inner surface of the ventricle. In each ventricle the terminal ramifications run back toward the base of the heart beneath the endocardium of the lateral wall, reaching as far as the auriculoventricular ring.

The spread of the contraction in the auricles is comparatively slow and wave-like, and by the time that P has risen to its peak and returned to the base line the contraction has spread to involve the whole of the auricular mass. Relaxation does not occur for some time—not until from .16 to .20 sec. after P has begun.

The delay in the passage of the impulse from auricles to ventricles seems to be at the auriculoventricular node. The tissues of the auriculoventricular bundle and its ramifications in either ventricle (the finer branches are known as Purkinje tissue) conduct the impulse very rapidly to the whole of the inner surface of the two ventricles, and the contraction spreads radially from the endocardial surface through the muscle of the ventricular wall to the pericardial surface at a much slower rate. The subpericardial layers are the last to be involved in the contraction and those fibers near the base of the left ventricle are the last of these. The ventricular muscle enters into contraction very rapidly, considering its size, because it is stimulated almost simultaneously at so many widely separated points by means of this complicated conduction mechanism.

During the spreading of the contraction throughout the ventricles the Q R S group is inscribed; and when it is completed all the ventricular fibers have become involved. Owing to the very effective method of distributing the contraction stimulus to the ventricles, the spreading of the contraction through them takes just about the same time as is needed for the spread of the contraction in the much smaller auricles. It is also a result of the way the contraction spreads in the ventricles that the electrical force produced at successive instants during the Q R S group repeatedly changes its direction in relation to the lead. The movement may be first downward, then upward and then down again as the electrical production within the muscle is predominantly first in one and then another direction.[1]

At the completion of the Q R S group with the whole of the ventricular muscle contracting, the line of the record rests at or very close to zero. There is an approximate electrical balance at this time due to the coincident equal activity in all parts of the ventricles. The T wave begins directly after this. It rises slowly during the time when the intraventricular pressure is rising, reaches its peak at about the same time as the pressure curve, and then begins to fall with the fall of the pressure. It ends with the onset of that sharp drop in the pressure which produces the closure of the aortic valves. Thus the beginning of the second heart sound comes with or just after the end of the T wave, just as it returns to the base line.

From its position in the heart cycle the T wave must be connected either with the full development of the ventricular contraction, or with the beginning of ventricular relaxation, or possibly with both of these. The generally accepted theory is that the T wave results from a disturbance of the electrical balance which follows the Q R S group, by the relaxation of certain fibers before others. The electrical effect of these fibers is removed from the electrical balance caused by synchronous contraction of the whole of the ventricular mass, and the T deflection results. By this theory the normal T wave indicates that relaxation affects the left and apical region of the ventricles before the right and basal region (Chap. IX). It is of course possible that some of the muscle fibers may relax as early as the beginning of this wave and yet not cause enough diminution in the contraction of the ventricular wall to lead to a fall in pressure within; but definite proof of this is as yet lacking.

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[1] Attention should be centered on the fact that it is the electrical potential that changes its direction during the Q R S group. We cannot properly conclude from this that the spreading of the contraction takes different directions.

There are several features which seem rather to favor the theory of a relation between the T wave and that feature of the ventricular contraction which determines a strong and efficient systole. These are the relation of the T wave to the rising intraventricular pressure, also the large size of the T wave after exercise, and its small size when the myocardium is weakened by disease or fatigue. The T wave may be said to represent the metabolism of the contraction, and its size to be due to the summated effects of all of the contracting fibers. By this theory the normal wave indicates that the ventricular contraction predominates in the right and basal region of the ventricles.

So far as knowledge goes as opposed to hypothesis a correct understanding of the T wave has not been achieved. The two theories presented are both unproved, though I favor the second as does Einthoven. Not the least weighty arguments in its favor, I feel, are the variations shown by this wave in health and disease as they will be detailed in the later chapters.

SUMMARY

The electrocardiogram is due to the electrical production coincident with the contraction and relaxation of the muscles of the different chambers of the heart.

The P wave is due to the electrical production during the spreading of the contraction over the auricles.

The Q R S group of waves is due to the electrical production during the spreading of the contraction over the ventricles.

The T wave is due to the electrical production during the latter part of the ventricle systole, and may be due either to the coincident contraction of all the muscle fibers of the ventricles or to the fact that certain of these fibers relax before others, or to a combination of these two processes.

Chapter II

THE NORMAL ELECTROCARDIOGRAM

We have considered as much of the theory of the electrocardiogram as it is necessary for the physician to have in mind, unless he intends to do more than simply apply his knowledge of the electrocardiogram to the diagnosis of the diseased hearts which he may encounter. The waves of the electrocardiogram have been definitely connected with those processes of the auricular and ventricular contractions which cause them, and it has been pointed out that the same wave has a different height and form in the different leads. The height of any wave will be different in the different leads of a record and usually will be largest in one of them, not so large in the other two. There are times when the excursion may be equally large in two leads as is the case with P of Figure 5 A, T of Figure 5 G and R of Figure 5 G and H.

The size of the largest excursion of any wave, no matter in which lead this may be recorded, is an indication of the voltage within the heart giving rise to that wave. The differing heights of the same wave in the three leads depend upon the direction within the heart of the current producing that wave and how favorably the leads are situated for recording this current. Favorable leads will give a good representation of the voltage of a wave. Unfavorable leads will give but a small excursion no matter how large the voltage. A lead may be favorable for one wave of a record and not for the others. Thus in Figure 5 A Lead 3 is unfavorable for P but favorable for Q R S and for T, while in record G Lead 3 is unfavorable for Q R S and for T but not for P. In Figure 6 Lead 3 is favorable for Q R S but not for P or for T. The variable *direction* of a wave in the three leads depends upon the relation of the direction of the heart's current to the direction of that lead, hence the same current may cause an upward deflection in one lead and a downward in another, e.g., the T wave of Figure 5 A.

The *voltage* governs the size of the largest deflection in whatever lead it may be, while the *direction* of the current in the heart determines the relative size of the wave in different leads and whether it is upward or downward in each. The reader will realize that the height which a wave shows in its largest lead is a better measure of the electrical potential of that part of the heart cycle than the height of this wave in smaller leads. These features are considered in detail in Chapter IX.

In examining an electrocardiographic record there are four features of each wave which must always be noted:

1. The maximum height recorded in any lead (voltage).

2. The relation of the height and direction of the three views of the wave obtained by the three leads.

3. The form of the wave or peak—whether sharp, rounded, notched, etc.

4. The duration as measured by its straddle on the base line of the record.

The height of a wave should be measured from the zero level of the record, either upward or downward according to the direction of the wave. When measuring upward we should start from the top of the shadow of the string and measure to the top of the deflection; when measuring downward we should start from the bottom of the shadow of the string and measure to the lowest point of the deflection. In measuring the duration of a wave it is well to find one which leaves the base line synchronous with one of the time lines. We may then count the number of time lines which occur before the deflection returns again to the zero level. The wave should be measured in all three leads and the largest measurement of its duration should be accepted, for sometimes either the beginning or the end of a wave may be unfavorably recorded in one of the leads.

The variations in the waves of the eight different records of Figure 5 are typical of those which occur in records from different normal hearts. They depend upon slight differences

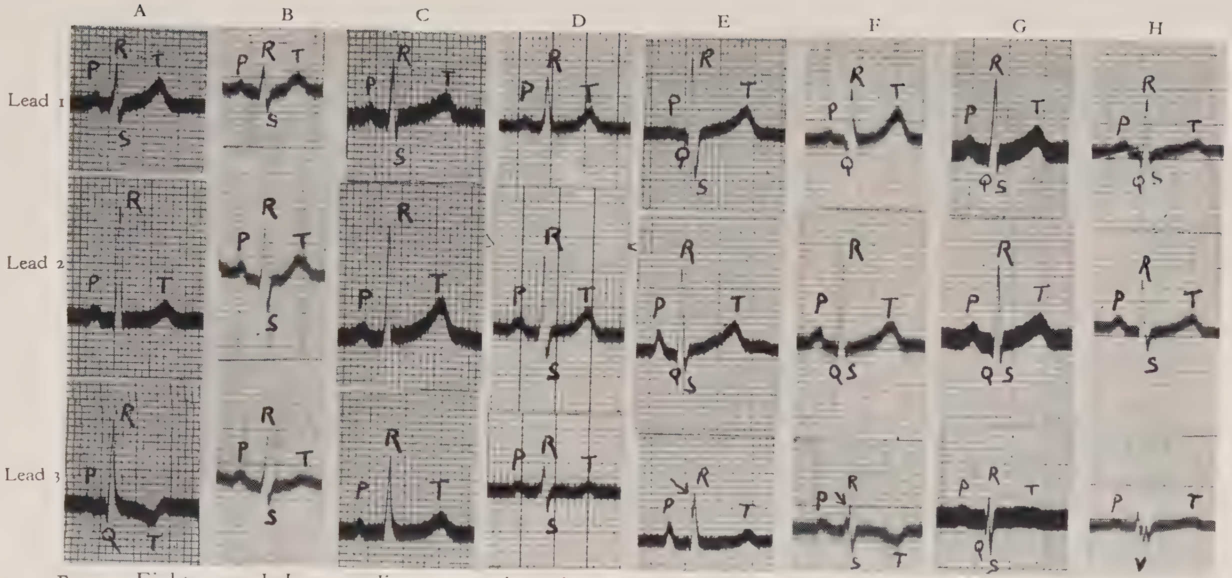

FIG. 5. Eight normal electrocardiograms to show the variations which may be encountered. In each case the upper electrocardiogram is by Lead 1, the central one by Lead 2 and the lower by Lead 3.

Note the variations in the relative height of R in Leads 1, 2 and 3 as we pass from Record A to Record H. Lead 2 always has the largest value, Lead 1 and Lead 3 vary reciprocally.

The fine vibrations of the line of the record, best seen in Lead 1 of B and C, are due to the electrical activity of the striate muscle of the extremities. The patient was not sufficiently relaxed. The blurring of the line of the record in B, F and H is due to the string shadow not being sharply focused.

The arrows in Lead 3 of Records E and F point to a notching or slurring of R. In Record H, Lead 3 has the Q R S group of the vibratory type (v).

Records B, F and H show a system of time lines produced by a different type of recorder. In these, and all subsequent records like them, the time from the left of each pair of vertical lines to the left of the next pair is .20 sec.

in the manner in which the contraction involves the muscle fibers of the auricles and ventricles. The differences in the P wave and in the Q R S group depend in part upon differences in the relative size and position of the muscle masses of various parts of the auricles or ventricles. The variations of the Q R S group depend chiefly upon slight differences in the path of the contraction wave in the ventricles, due to individual variations in the terminal branchings of the auriculoventricular bundle. The variations of P are probably very little due to variations in the path of the contraction wave because of the diffuse radial spreading of the contraction through the auricular muscle from the sinus node. It is probably for this reason that the P wave shows less variation than the Q R S group in normal hearts. The variations of T in the three leads are due to variations in the late phases of ventricular systole and may be explained according to either of the theories of the cause of this wave. According to the theory favored by the author, the process of contraction which causes the T wave may have more or less activity, causing a larger or a smaller wave, and the direction of T may vary because the process predominates in different parts of the ventricular muscle in different hearts. According to the other theory we may say that the early relaxation of ventricular fibers involves a greater or a lesser number, thus causing a larger or a smaller wave, and that the direction varies because the relaxation begins in different parts of the ventricular muscle in different hearts.

Since the normal variations are so numerous and so marked, it is necessary for the clinician to appreciate very clearly their limits. This knowledge has been accumulated slowly because of the difficulty of being certain that a given heart is surely normal. Still, by experience in correlating the electrocardiogram with the symptoms and physical signs in a large number of normal and abnormal individuals, considering also their clinical progress and a certain amount of autopsy material, it has become possible to make quite definite statements as to the characteristics of the waves of a normal electrocardiogram. When waves are found which do not agree with these standards we are able to predict that the contraction which produces them is in some way abnormal. As has been stated, each wave can vary in several respects, and it is by considering carefully the normal limits of each sort of variation that we determine whether or not the wave is a normal one. If it is not, we can usually tell from the character of the abnormality whether this is due to abnormal function or to muscle disease.

The Normal P Wave

The P wave is a rounded elevation whose height should be between 1 and 2 mm. in the lead which shows its largest excursion. Its voltage is a rough index of the auricular function, a small P meaning a poorly functioning muscle and a large one, a muscle which is functioning well. If the height exceeds the limit of 2 mm., then we must consider that the muscle is overactive or hypertrophied (Chap. III).

P is usually largest in Lead 2, though sometimes P 1 or P 3 may equal it,[1] in which case the other of these two has no excursion at all or may vary slightly above or below the zero level, as in Records A and E of Figure 5. The P wave of normal hearts is not frankly downward in any of the three leads, though it may be diphasic, having both upward and downward deflections, in Lead 3 (Fig. 5 A) or more rarely in Lead 1.

In about a third of all records from normal hearts the P wave will show a notching or doubling of its peak, as in Records A and F of Figure 5. This usually appears in Leads 2 and 3, but perhaps only in Lead 2. In Lead 1 it is rare, but should probably not be considered an abnormal finding. At one time notching of P was considered to indicate auricular hypertrophy. However, in a very careful study by Lewis and Gilder, of a group of 52 young men from whom possible abnormals were most carefully excluded, notching of P occurred in one or more leads in 17 records, P 2 was notched seventeen times, P 3 ten times and P 1 twice. This agrees with the findings in a smaller group of normal college students who were examined by the author. In this group of 26 students P was notched in one or more leads nine times. P 2 was notched seven times, P 3 seven times and P 1 four times. It can be admitted that notching of P might occur because of an occult auricular abnormality in a small number of cases, but it is scarcely possible that 33 per cent of a group of supposedly normal hearts should have an undiscoverable abnormality. It seems that this notching must be accepted as one of the normal variations of the P wave, and Lewis has also considered it so.

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[1] P 1 means the P wave of Lead 1, P 2 the P wave of Lead 2, and so always when the waves are referred to by a letter followed by a number.

The cause of notching of P is not definitely known, but it may be suggested as a working hypothesis that the normal P wave is composed of two overlapping electrical effects, one of which is due to each auricle. In the majority of instances these peaks fall so close to one another that no notching occurs. Occasionally the path of the contraction in one auricle from the sinus node to the tip of the appendix or the base of the auricle may be longer than usual, so that the potential of that side will be slightly delayed in reaching its height. Thus the peaks of the two electrical effects might not coincide as closely as usual and a notching of P would result. This hypothesis will help to "explain" some of the abnormal variations of P and will be mentioned further when they are discussed.

Duration of P. The duration of the P wave, the width of its straddle measured on the base line, is usually .08 sec. In 10 of the author's series of 26 normal students it was found to be between .08 and .10 sec., so the latter figure probably should not be considered abnormal. In one case the time was .06 sec. This feature of the P wave indicates the time necessary for the contraction to spread over the whole of the auricular muscle and so must be reckoned from the lead in which it is longest—usually Lead 2. We do not know the factors which make this time unusually short, but it is generally found so with rapid heart rates and in children. Anything increasing the length of the path of the contraction would be expected to lengthen this time; and it is also conceivable that a lowered functional condition (depression of conductivity) might prolong it.

If the P wave is normal in all these respects—its height, its direction in the three leads, its form and its duration—we can conclude that the auricular contraction has passed in a normal way through a normal auricular muscle.

The P-R Level

From the end of the P wave until the beginning of the Q R S group the line of the record rests very near to zero, but rarely stays directly upon it in all three leads. In the author's normal series this deflection was downward in all three leads in 12 records and downward in two leads in 12 records. The 2 remaining records showed no deflection in any lead. There is at present no clinical significance attached to the deflection during this interval, though I have seen it occasionally as great as 2.5 mm. It is usually large when the P wave is large, though having an opposite direction. It is due to auricular activity and is the auricular analogue of the T wave of the ventricular beats, recording the potential caused by the contraction of the auricles after this process has spread to involve the whole of the auricular muscle. In the heart-block records of Figure 34 D and 35 A it can be seen that the descending limb of P goes slightly below the zero level, and remains there for 0.18 sec. This deflection has been called the auricular T wave.

The P-R Interval

There is an interval of about 0.16 sec. from the beginning of the P wave until the Q R S group begins. This space between the beginning of the electrical effects of the auricular and ventricular contractions is the measure of the time taken by the contraction-producing impulse in passing from the sinus node to the ventricles. It is the auriculoventricular conduction time and is called the P-R interval, rather loosely, for the important thing is the time before the Q R S group begins, irrespective of whether Q, R or S is the first deflection of the group. This interval is not usually the same in all three leads from the same person, because one lead may be unfavorable for recording the beginning of the P wave or of Q R S (Chap. IX). Since the interval is an index of the auriculoventricular conduction time it is obvious that the longest P-R interval which appears in any of the leads will be the most correct measurement, and this is usually found in Lead 2.

The P-R interval varies with the heart rate, being shorter with more rapid rates and longer with slower ones. It also tends to be shorter in children than in adults. It may be as short as .12 sec. or as long as .20 sec. without being considered abnormal, unless it should be found to be at or near this maximum in a child, or in an adult with a heart rate of 90 or over. The normal variations in the A-V conduction time are dependent upon the functional condition of the conducting system, short conduction time being dependent upon good functional condition and a low grade of vagus activity, while prolonged conduction may be due to a poor condition of the bundle tissues or excessive activity of the vagus.

The Q R S Group

The Q R S group is subject to an infinite number of variations, because the three peaks can each vary in height more or less independently of the others. When examining this group of waves we must attend particularly to: (1) The relative height and direction of the largest wave in each of the three leads; (2) the presence and character of notching of the waves; (3) the height of the largest deflection in any of the leads (voltage of Q R S); (4) the duration of the Q R S group as measured by its width on the base line.

In all three leads of a normal record there will be an upward peak called R (Fig. 5). It may be relatively small in Lead 1 or Lead 3, but it always reaches a height in Lead 2 not exceeded in any other. From such a series of records as Figure 5 we can see that as the height of R 1 diminishes in relation to R 2 so does the relative height of R 3 increase.

Follow the series of records E, D, C, B, A; R 2 is plainly larger than R 3 in record E, while in record A these have become more nearly equal in size and R 1 is small in relation to R 2. Likewise, as the height of R 3 decreases in relation to R 2 in the series C, D, E, F, G, so does the relative height of R 1 increase. R 1 and R 3 are seen to be reciprocal, and in this we can discern the influence of Einthoven's law of the leads which is discussed in Chapter IX:

(Excursion Lead 1 + Excursion Lead 3 = Excursion Lead 2).

The variations of the waves Q and S bear a certain relation to the relative heights of R 1, R 2, and R 3. Curves with relatively small R 1 tend to have a deeper S 1 than do curves of the other type and to have S 3 small or absent. Q is nearly always best developed in Lead 3 and absent in Lead 1. Conversely, those curves with relatively small R 3 usually show the S wave better developed in Lead 3 than in Lead 1, while Q is better developed in Lead 1 than in Lead 3. These things will appear more plainly if the Q and S waves of Records A, B and C are compared with the same waves of Records E, F, G and H.

When R has a relatively small excursion in Lead 3 it is common for the Q R S group in that lead to have *only* small excursions, so that it is rather a vibratory complex, as in records G and H. Such vibratory complexes are a rare occurrence in Lead 1, even when the value of R in that lead is relatively small. They are occasionally found, but are not a normal occurrence.

These variations in Q R S are caused by variations in the direction of the current within the heart and by the relation of the three leads to this current (Chap. IX). There is, however, considerable clinical interest in this division of the normal curves into two general groups as indicated (those with *relatively* small R 1 and large R 3 and those with *relatively* small R 3 and large R 1) because of its relation to the diagnosis of ventricular hypertrophy (Chap. III). Three factors combine their influence in producing these variations in Q R S and sometimes one, sometimes another, may be of predominant importance. The factors are: (1) The normal variations in the structure and distribution of the terminal arborizations of the auriculoventricular bundle inside the two ventricles; (2) the position of the heart within the thorax, whether transverse or vertical; (3) the relation of the weight of the muscle masses of the right and left ventricles.

If the ventricles are separated and weighed it is found that the left ventricle is heavier than the right and that normal hearts give as an average proportional weight: left is to right as 1.8 is to 1. The normal variations lie between 1.5 to 1, and 2.15 to 1. These variations in the weight of the ventricles of normal hearts help to determine the variations in the Q R S group of the normal electrocardiogram. Hearts in which the right ventricle is relatively more heavy than the average, though the L/R ratio is still within normal limits, tend to produce curves like A and B of the figure with relatively small R 1 and large R 3, usually with an S wave in Lead 1 and a Q in Lead 3. Hearts with the left ventricle relatively more heavy than the average, though again not outside the normal limits, tend to produce curves, like F, G and H of the figure with relatively small R 3 and large R 1, the S wave being usually most marked in Lead 3 and Q in Lead 1, or perhaps with a vibratory Q R S in Lead 3.

This is not a matter of the actual weight of each ventricle, but of their relative weights, for normal electrocardiograms like C, D and E result when the left is heavier than the right. If the proportion of L to R as 1.8 to 1 be maintained, the heart will tend to give a Q R S group within the normal limits shown in Figure 5, whether the actual ventricular weights are 54 and 30 gm. respectively or 108 and 60 gm. The feature influencing the variations in Q R S is the relation of the mass of one ventricle to that of the other, and not the actual size of either.

The position of the heart within the chest, whether tending to have its long axis more transverse or more vertical, has a great influence in modifying the Q R S group. This is especially so with the normal heart, less so with hearts which have hypertrophied ventricles. The effect of a transverse position of the heart as it lies upon a high diaphragm is similar to that of a relatively heavier left ventricle, so that R tends to be smallest in Lead 3 and records of the type of F, G and H of Figure 5 are likely to be obtained. The effect of a vertical position of the heart—the so-called "drop heart"—is the same as that of a relatively heavier right ventricle with the R wave smallest in Lead 1 and records like A and B of the figure are usual.

Probably the most important cause of variations in the Q R S groups of *normal* hearts is the variable distribution—the "architecture," as Wilson and Herrman have called it—of the terminal arborizations of the Purkinje system within the ventricles. These variations result in the stimulus coming to the various parts of the ventricles in a different order in different hearts and that part of the electrocardiogram, Q R S, due to the spreading of the contraction is thereby affected. If the path to the right ventricle is shorter than usual the electrical effects of this ventricle will start to develop their full strength earlier than usual and will be more prominent during the Q R S group, so that the curves approaching the right ventricular type are likely to appear (A, B and C of Fig. 5). If the path to the left ventricle is shorter, this ventricle will impress its signs upon the Q R S group and we shall be more likely to find the left ventricular type of curve (F, G and H of Fig. 5).

All three of these factors are concerned in the form of each Q R S group, so that we must bear each one in mind as a possible cause for such unusual variations as may be encountered.

Notching of the Q R S Group. Notching of the R wave is especially prone to occur in Lead 3 when R is relatively small in this lead, and R 1 is about equal to R 2 (Fig. 5 F and H). The Q R S of Lead 3 in these records is often composed of a series of small vibrations, as in Record H of the figure, so that it is difficult or impossible to name the individual peaks. Notching of Q R S is occasionally seen in Lead 1 when the excursions are relatively small in this lead and R 2 and R 3 about equal in size, but it is rare to see anything like the vibratory or splintered wave groups which are so common in Lead 3. If they are found in Lead 1 the record cannot be considered a normal one.

If the process which causes notching occurs during the upward or downward limb of a large wave, it is as though the notch were stretched out and it may be changed to a slurring or thickening of one limb of R or S, as in Lead 3 of Figure 5 E. This has the same significance as notching, but must be clearly distinguished from the slurring so frequently seen as the R or S waves leave or approach the base line (Lead 3 of Figs. 5, A and C, and Lead 1 of Fig. 5 A) which does not have this significance.

When the notching occurs at the peak of a wave it results in a broadening or thickening of the peak which may amount to a true notch, as in Figure 5 F Lead 3. The normal thickening of the line as it changes direction at the peak of R or S must be distinguished as different from a notching.

Notching or slurring of the waves of the Q R S group can be considered a normal phenomenon when it is found in only one lead, and that lead one with a *relatively* small excursion of Q R S. If notching or slurring is found in two leads, it can only be considered normal when it occurs at the beginning or end of the Q R S group and very near to the base line. It is never normal to find notching in three leads, or near the peak of R in a lead of *relatively* large excursion.[1]

Notching is such an evident feature of abnormal electrocardiograms that it is necessary to realize that it may sometimes be normal. Its basis in the changing potential produced during the ventricular contraction is discussed in Chapter IX, and also what this may mean when referred to muscle function.

[1] On reviewing a normal series of 78 persons, 52 recorded by Lewis and 26 by the author, an interesting fact appears in regard to the notching of Q R S. Whereas T 3 is turned downward in 17 records, there are 13 of these which also have notching or splintering of the Q R S group in Lead 3. Moreover, of the 32 cases in the series with notching or splintering of Q R S, a downward T 3 is found in 13 (40 per cent) a frequency about double that of a downward T 3 in the series as a whole, i.e., 17 times in 78 cases, or 22 per cent. It seems evident that whatever leads to a notching of Q R S in Lead 3 tends also to produce a downward T wave in this lead.

The Size of Q R S. It has been stated that the voltage of a wave is approximately measured by the size of the deflection in the lead giving this wave its largest value. In Figure 5, then, the voltage of the Q R S group would be best shown by R 2 in all records but H, and in this record by both R 1 and R 2. In this figure the value for Q R S varies from 17 mm. to 8 mm. in different records.

In Lewis' series of 52 normal students the variations in the height of the largest wave of Q R S were from 5.5 mm. to 16.5 mm. with an average of 11 mm. In my own normal series the height varied from 8 mm. to 23.5 mm.; but these were college students and engaged in a certain amount of competitive athletics, while Lewis' series were medical students who presumably were older and not so much concerned with athletics. It is probable that the figures at either extreme should not be considered as normal, and so from a study of the frequency of occurrence of the different values in the combined series of 78 normal persons, it seems proper to set a minimum value of 7 mm. and a maximum value of 16 mm. as the normal limits for the voltage of Q R S recorded in its largest lead. The average for the combined series was 12 mm.

Whenever Lead 1 or Lead 3 gives Q R S a very small relative value the highest recorded value should properly be increased by 10 or 15 per cent to get a truer measure of its voltage. The mathematical reasons for this are explained in Chapter IX.[1]

Variations in the voltage of Q R S occur from time to time in normal persons and frequently in patients with cardiac failure. Provided that the Q R S group does not change its form in any other way, variations in its voltage can usually be shown to be coincident with variations in the nutritional state of the heart muscle. Small values for R seem to mean a muscle which is below par, perhaps through lack of exercise or perhaps through some fundamental disturbances in its nutrition, while large values mean a strong well-nourished muscle. During convalescence from an acute disease, for instance, the value of R increases as it does also when the condition of the circulation improves after cardiac failure. The differences noted between the voltage of Q R S in the two series of normals just mentioned are in line with this hypothesis, the more sedentary group of Lewis showing smaller values than the more athletic group of the author.

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[1] The value of R, as recorded in its highest lead, differs from its voltage more and more as the direction of its potential within the body is more nearly perpendicular to the line of any lead, the recorded value being only 87 per cent of the voltage when the direction is exactly perpendicular to a lead. An approximately perpendicular angle for the Q R S group may be recognized from the three leads of the record by the fact that in one lead there is a very small *relative* value for R, while in the other leads the values are larger and about equal, as in Records A and H of Figure 5. It is in such records that the maximum recorded value of R should be increased by 10 or 15 per cent in order to get a truer measure of the current from the heart.

Duration of Q R S. Normal hearts show differences in the duration of the Q R S group. The time from the beginning of Q or of R, whichever is the first to develop, to the end of R or S, whichever finishes the group, is the time consumed by the spreading of the contraction throughout the ventricles: in other words, the time from the first ventricular activity until the contraction has come to involve the whole ventricular musculature. The longest time measurable in any one of the three leads gives its correct measurement, and in normal hearts this time is found to vary between .06 and .10 sec.

The fact that children's hearts always have a very brief Q R S suggests that a short auriculoventricular conduction system and a short path through the ventricular muscle, when taken together, even if not separately, will give a brief Q R S group. There are other factors, though, for the same heart will have a shorter Q R S interval when beating rapidly than when beating slowly. Our experience with hearts showing hypertrophy (Chap. III, p. 41) teaches us that one factor which prolongs this time of spreading contraction is an increased thickness of the wall of the left ventricle. It is likely that variations in the functional condition of the A-V bundle and its branches, or perhaps even of the ventricular muscle, will shorten or lengthen the duration of Q R S.

The T Wave

The T wave is the last of the ventricular waves. It has several variable features which should be noticed: (1) Its direction in the three leads, whether upward or downward; (2) its maximum excursion; (3) the interval before its rise; (4) its duration.

Direction of the T Wave. In records from normal hearts the T wave is always directed upward in Leads 1 and 2, though the size in either of these leads may be small in relation to the maximum shown in the other. About 1/4 or 1/5 of the records from normal hearts have T turned downward in Lead 3, as in Figure 5, A and F, while the remainder show an upward T 3. A downward T 3 may at times be due to a high position of the diaphragm. This would tip the apex of the heart upward and would tend to produce a relatively small or even inverted T 3 just as it tends to produce a small or inverted R 3. A person with a long narrow chest would, for a similar reason, tend to have T relatively small in Lead 1. As with the Q R S group, however, the varying position of the heart is only one of the causes of variations in the direction and height of the T waves.

Variations in the direction within the heart of the electrical potential giving rise to T are the principal reason why the T waves differ. Sometimes the direction is such as to produce a relatively small T 1, sometimes a relatively small T 3 and sometimes a downward T 3. The direction of the potential within the heart will be modified as described by the position of the heart in the body.

We do not at present know why the current of this part of the heartbeat varies in direction. It is suggested by the changes in the T wave which accompany ventricular hypertrophy (Chap. III, p. 41) that an inverted T 3 may result from the influence of predominance of the right ventricle during the contraction.[1] Slight precedence of the contraction of a normal right ventricle might, then, explain the downward T 3 which is found in certain normal records.

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[1] This idea receives some support from the following: In the author's normal series there are 6 records with downward T 3, 4 of which have a Q R S in the third lead like that of Figure 5 A, with Q, a large R and no S wave. In Lewis' normal series there are 10 records with downward T 3 and 5 of these

Either of the two theories of the causation of T may be applied fairly successfully as an "explanation" of its variable direction in Lead 3, but it is only pushing our ignorance behind us to feel that they do explain. For example, by one theory we would say that the process of contraction which causes the T wave predominates in different regions of different hearts, thus producing the variation in the direction of the electrical potential. By the other theory we would say that the early relaxation of muscle fibers that causes the T wave does not occur in quite the same region of each heart and therefore the direction of the electrical potential is different.

In spite of this apparent vagueness we know by experience and by experiment that the T wave is found as described in normal hearts and not otherwise. If the heart is experimentally injured the T wave will be changed.

Height of T. The height of T in the lead showing the largest excursion does not exceed 5 mm. normally, nor should it show less than 2.0 mm. When the T wave gives less than 2.0 mm. it should probably be considered abnormal. The average height in the combined normal series of Lewis and the author was 3 mm. The height of the T wave must be increased by 10 or 15 per cent, just as with the R wave, if any one lead should show a very small relative deflection.

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have a similar Q R S in that lead. This Q R S is like that found in the third lead with right axis deviation of Q R S, and moreover, 5 of the 9 records showing it have a well-marked S 1 and a relatively small R 1, so that S approaches R in size in this lead if it does not equal it. This is a further suggestion of right ventricular activity and might be due to the right ventricle contracting slightly ahead of its usual time. This might result from an unusually short path along the branch of the auriculoventricular bundle to the right ventricle, or an unusually long path to the left ventricle. The other 7 records of the combined series that have T 3 directed downward show, like Figure 5 F, a relatively small notched Q R S in Lead 3, while the relative size of R in the other leads suggests a left axis deviation of Q R S. In spite of this all but one of them have an S wave of from 2 mm. to 4 mm. in Lead 1, which wave is considered to be a function of the right ventricle. This tendency to a small Q R S in Lead 3 with downward direction of T 3 could result from a transverse position of the heart because of a high diaphragm.

The physiological basis of variations in the voltage of T is not definitely known. It seems to vary roughly with the strength of the cardiac contraction, being large in the hearts of athletes and, in the same person, becoming larger after exercise. The variation in the voltage of T does not parallel that of the Q R S group from case to case except in the most general way. An R wave toward the upper limit of normal may be seen in a normal person who has a small T wave, and vice versa. In general, however, a large voltage of Q R S does go with a large voltage of T, a fact in accord with the theories given for their variations. If the R wave represents the nutritional state of the muscle and the T the strength or intensity of the contraction, it is to be expected that they would tend to vary together; but it can be understood how one or the other might vary independently.

It must be emphasized again that what has been said about the height of the Q R S and the T waves is applicable only to records which arise by a normal process of contraction. Abnormal modes of contraction in themselves can vary the height of these waves, as is pointed out in Chapter IV, so that if the Q R S group is notched or slurred the height of Q R S and of T is likely to be different from what it would have been without the notching.

The S-T Interval

The interval after the Q R S group, and before the onset of the rise or fall of the first part of T, has been described as one of slight or no deflection. The reason for this has been said to be that there is a balance of potential at this time, because all parts of the ventricles are in the same state of contraction. Neither this explanation nor the thing which it designs to explain is more than approximately correct, for it is plain that the state of contraction of a fiber group which was stimulated at the beginning of the Q R S complex will be further advanced than that of one which was stimulated .08 sec. later at the end of this group. Likewise a careful scrutiny of this portion of the electrocardiogram will reveal that the line of the record is rarely exactly upon the zero level, but is a few tenths of a millimeter above or below it, and that, as is usual, the deflection is different in different leads. The size of this deflection in normal records is never over 1 mm. and not usually as much as this.

There is then, at this time, a very slight production of electrical potential within the heart. In the author's series (Lewis unfortunately did not mention this interval in his study of normals) it usually gives an upward deflection in two leads or in three (16 cases) though it may be downward in one or more leads (7 cases). In 3 cases it was at zero in all three leads.

The line may remain at this level in one or two leads for as much as .12 or .16 sec., but more usually after .02 or .04 sec. it curves gradually upward or downward toward the peak of the T wave. In most cases it does not pause for even this length of time in more than one lead.

It is important to draw attention to this part of the ventricular complex, for it varies greatly in certain pathological cases and also under the influence of digitalis. It sometimes varies when the variation in the T wave itself is less pronounced or is of a different character.

Duration of the T Wave. The duration of T, from the end of the Q R S group to the end of T, has been considered by some to be an indication of the duration of the ventricular systole. This is incorrect, because of the variations which may occur in the duration of Q R S, or at least it is less correct than to consider the total duration of the ventricular complex as a guide to the duration of systole. From the very beginning of the Q R S group there are more and more muscle fibers entering into contraction, until, at its end, all are contracting. Even the intraventricular pressure begins to rise before the end of the Q R S group is reached (Fig. 3) so that it seems quite illogical to consider the duration of T as being the duration of systole. It is true that the total duration of the ventricular complex is greater than that of the effective systolic activity, the mechanical effect of contraction, but this relation is probably more constant than that between the duration of T and the mechanical effect.

The duration of the systole, from the beginning of Q R S to the end of T, must be determined from the lead in which it is longest, and other things being equal, it will depend upon the heart rate. In my series of normals the heart rates and duration of the ventricular complex appeared as follows:

Duration of Ventricular Complex

Rate		Average	Low	High
52	1 case	.46	...	...
60 to 69	8 cases	.40	.36	.42
70 to 79	10 cases	.375	.34	.40
80 to 89	4 cases	.355	.34	.36
90 to 99	3 cases	.34	.34	.34

This series is too small to enable us to draw far-reaching conclusions, but it appears that if with a heart rate of 70 or more the duration of the ventricular complex should be .42 sec. or more, we could not consider the heart action normal.

There is good evidence from experimental work that the duration of systole will be increased with increased diastolic filling of the ventricles, even if the rate remains unchanged. It is possible that the relation between heart rate and the duration of the ventricular complex may thus express the degree of cardiac dilatation at the various rates, but this is as yet clinically unproved.

The state of cardiac nutrition has been suggested as a possible cause of the variations in the duration of systole, poor nutrition causing prolongation, and vice versa. This theory is not fully accepted as yet, though there are some observations which seem to support it.

SUMMARY

	P	QRS	T
Height in largest lead	1 to 2 mm.	7 to 16 mm.	1 to 5 mm.
Direction			
Lead 1	Upward	Chiefly upward (R). Q or S may be present either singly or together but neither as large as R.	Upward
Lead 2	Upward	Chiefly upward (R). Q or S may be present either singly or together but neither as large as R.	Upward
Lead 3	Upward, diphasic or downward.	Q, S or both may equal but not exceed R. Q R S group may be of vibratory type.	Upward, diphasic or downward.
Form	Rounded; may be notched.	One, two or three sharply pointed peaks, or vibratory group. A peak may be notched in a lead of relatively small excursion or near base line.	Peaked
Duration	Not more than .10 sec.	Not more than .10 sec.	Q R S-T varies with heart rate .32 to .42 sec.

P-R interval varies with heart rate .12 to .20 sec.

Chapter III

HYPERTROPHY OF THE CHAMBERS OF THE HEART

Of cardiac abnormalities which may be determined from the electrocardiogram, the most frequent is hypertrophy of one or another chamber. Hypertrophy of the auricular or of the ventricular muscle is not to be considered as evidence of disease of the myocardium, for it may arise from purely mechanical causes which demand an increased propulsion of blood by the affected chamber. As examples of this, hypertrophy of the left ventricle may be caused by high blood-pressure or disease of the aortic valve, and right ventricular hypertrophy by congenital narrowing of the pulmonary artery or increased pressure in the pulmonary circuit due to mitral disease. Besides the mechanical causes of hypertrophy, there is what might be called a pathological cause. If the muscle of a ventricle is diffusely affected by disease its power to propel blood will fail; blood will accumulate within it, the demand upon it will be thereby increased and a hypertrophy will result. The electrocardiogram of such a heart will be affected by the hypertrophy, but also by the disease, so that it is usually possible to distinguish from the record whether a heart is hypertrophied by purely mechanical causes and has a practically sound muscle, or whether disease of the muscle is the cause of the enlargement.

We shall consider in this chapter only the changes in the electrocardiogram which arise from hypertrophy of the muscle, and not those due to its disease.

Auricular Hypertrophy

Hypertrophy of one or the other auricle cannot be diagnosed from the electrocardiogram. It is not likely that both auricles are always equally hypertrophied, but for the present we cannot distinguish between the effects of the right and the left chamber upon the P wave.

There have been no correlated pathological and electrocardiographic studies of the auricles and the P wave, but it is a common pathological observation that, in hearts that have mitral stenosis, auricular hypertrophy is extremely common, in fact, quite universal. We may then take the P wave of patients with mitral stenosis as exemplifying the curve of auricular hypertrophy, and we find that there are three features which are conspicuously frequent in such curves: (1) The height of P is excessive, being 2 mm. or more in the lead having the largest excursion in 75 per cent of any large group of records from hearts with mitral stenosis (Fig. 6 A and B). The wave is sometimes found as large as 5 mm. (2) The duration of P is excessive, being over .10 sec. in 85 per cent of the records (Fig. 6 A, B and C).

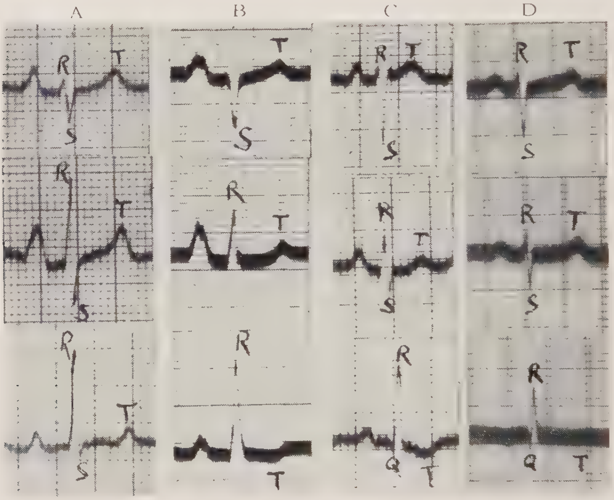

Fig. 6. Records from four different hearts to show varying degrees of right axis deviation of Q R S. Record A indicates the least degree and Record D the greatest. Note that S 1 increases in size in relation to R 3 as the degree of deviation increases. In Records C and D, S 2 shows a progressive increase in its size.

Records A and B each have a large and wide P wave, indicative of auricular hypertrophy. Record B has a plainly notched P wave in each of the three leads.

(3) Notching of P (Fig. 6 B) occurs with unusual frequency, being found in about 60 per cent of records from hearts with mitral stenosis, while in only about 30 per cent of records from supposed normals.

Since the P wave is due to muscle activity during the spreading of the contraction over the auricles, it is understandable on theoretical grounds that more electric potential would be produced when the muscle is increased in amount and that thus the height of P would be increased. It is also understandable that it would take longer for the contraction wave to spread throughout the greater mass of muscle, so that the duration of P, which represents the time occupied by this spreading, would be prolonged over the normal limits. The notching can be explained on the basis of the hypothesis mentioned in Chapter II, for if the auricles are unequally enlarged their potentials will be more likely to develop to a maximum at different times, causing a double peak or notching. It seems then that we are justified in considering *auricular hypertrophy* to be present when we find a *P wave of over* 2 *mm.*, that has a *duration of over* .10 *sec.* and that is *notched;* or if we find one that shows any two of these signs, particularly the first two.

Ventricular Hypertrophy

Hypertrophy of one or the other ventricle has a very evident effect upon the Q R S group, producing the changes which Lewis speaks of as a sign of right or left ventricular preponderance, meaning that the right or left ventricle is preponderantly hypertrophied. This use of the word preponderance has the fault of overstressing the importance of the weight of the ventricles. When the term was introduced, the ventricular weight ratio was considered to be the determining factor in the form taken by Q R S in the three leads. This has not proved to be uniformly the case. For this reason it seems better to express these variations of the Q R S group by some term which is referable only to this group. It is pointed out in Chapter IX that the direction of the waves of Q R S is determined by the varying direction of the electrical potentials within the heart during its occurrence.

The direction of the electrical axis of Q R S controls the direction of the largest excursion of Q R S in the three leads. This largest potential has been called by White the electrical axis and the direction of this axis can be determined roughly by referring to Figure 10 and its explanation. In records like those of Figure 5 with Q R S chiefly upward in all three leads (Lead 1 +, Lead 2 +, Lead 3 +) the electrical potential producing the peaks of the R waves has a direction within the heart which lies between 30° and 90°.

Hypertrophy of the right ventricle tends to change the direction of the electrical axis of Q R S toward the patient's right producing records like those of Figure 6 with the chief deflection of Q R S downward in Lead 1 and upward in Lead 3. The sector (Lead 1 − Lead 2 + Lead 3 +) in Figure 10 is to the patient's right of the sector for normal excursions. Hypertrophy of the left ventricle tends to change the direction of the electrical axis of Q R S toward the patient's left, giving such records as are seen in Figure 7 with the chief deflection of Q R S downward in Lead 3 and upward in Lead 1. The sector (Lead 1 +, Lead 2 +, Lead 3 −) in Figure 10 is to the left of the normal.

We may speak accordingly of a deviation of the electrical axis of Q R S toward the right or toward the left. It will be readily realized that the direction of the electrical axis may be affected by other things than hypertrophy of the right or left ventricle. Variations in the distribution of the branches of the auriculoventricular system and in the angle of inclination of the heart as it lies within the chest have been discovered to have an important influence upon Q R S, as has been discussed in the previous chapter.

Hypertrophy of the right ventricle only tends to produce records like those of Figure 6 when the left ventricle does not increase proportionately at the same time. The change is due to a predominance of the effect of one ventricle over that of the other. For example, a heart with ventricular weights of left = 158 gm. and right = 98 gm., the L/R ratio being

1.61 :1, showed an electrocardiogram with the Q R S group as follows:

Lead 1, R = 1 mm., S = 4 mm.
Lead 2, R = 6 mm., S = 3 mm.
Lead 3, R = 5 mm., S = 1 mm.

This is an example of slight right axis deviation of Q R S similar to Record A of Figure 6. Another heart with ventricular weights of left = 105 gm. and right = 128 gm., the L/R ratio being 0.82 : one gave a record with the Q R S group as follows:

Lead 1, R = 0.5 mm., S = 8 mm.
Lead 2, R = 8.0 mm., S = 5 mm.
Lead 3, R = 11.0 mm., S = 3 mm.

This is an example of moderate or marked right axis deviation similar to Records B or C of Figure 6.

Right axis deviation of Q R S is indicated when Lead 1 has no R wave, or but a small one, with S larger than R. At the same time R 3 is larger than R 2. If R and S are of the same height in Lead 1 with R 2 and R 3 equal it should be considered to indicate a condition just beyond the normal limit. Figure 6 shows four records, A, B, C and D with increasing degrees of right axis deviation. As the degree of this axis deviation increases S 1 shows an excursion which is an increasing percentage of the R wave of Lead 3 until it may equal R 3 or even exceed it, as in Records C and D of the figure. As S 1 becomes relatively larger, R 2 becomes a smaller fraction of the largest excursion of Q R S and S 2 tends to become larger. The Q wave is practically never found in Lead 1 of records showing right axis deviation. It is commonly found in Lead 3, and if present in Lead 2 also, will be larger in Lead 3 than in Lead 2.

Further right axis deviation of Q R S leads to an increase of S 1 and S 2 at the expense of R 1 and R 2 until eventually S 2 is found larger than R 2, as in Record D of the figure. Such a degree of deviation to the right has very rarely been found in my experience except in hearts with congenital defects or acquired narrowing of the pulmonary arteries.

Left axis deviation of Q R S is indicated when Lead 3 has no R wave, or but a small one, with S larger than R. At the same time R 1 is larger than R 2. In Figure 7 are four records showing increasing degrees of left axis deviation. It is seen that S 1 is often absent from these records, and that S 3 becomes a progressively larger percentage of the maximum value of Q R S as the deviation of the axis toward the left becomes more marked. This is analogous to what happens to S 1 with right axis deviation. Moreover, R 2 becomes relatively smaller and S 2 relatively deeper with increasing deviation toward the left, just as occurs with marked right axis deviation (Fig. 7 c and d). Such a Lead 2 might be part of a record showing either right or left axis deviation, and without the other leads we could not say which it was; for example, compare Lead 2 of Figures 6 c and 7 c. When S 3 gives the largest excursion of Q R S in any lead and S 2 is larger than R 1 we have an extreme grade of left axis deviation (Fig. 7 d).

Q is most apt to be present in Lead 1 of records which show left axis deviation. If it appears in more than one lead it will usually have its greatest value in Lead 1, rarely ever appearing in Lead 3.

These statements apply only to *records in which the Q R S group is normal except for the axis deviation.* Should the Q R S group have an abnormally long duration or should it show notching or thickening in more than one lead, then a preponderant hypertrophy of one or the other ventricle may cease to determine the form of Q R S. In such a case the whole spread of the contraction may be abnormal and the effect of this may completely overbalance the effect of the abnormal relation of ventricular weights (Chap. IV).

Voltage of Q R S. The height of the largest recorded wave of Q R S, in whatever lead this may be, varies in general with the degree of ventricular hypertrophy, being larger in large hearts and especially if a marked right or left axis deviation is shown. It is also, on the average, larger in *large* hearts which show a normal direction of the axis, than in hearts of normal size. Both of these statements are made with the qualification that the physiological condition of the muscle is equally good or equally poor in the hearts compared, and that there is no myocardial abnormality which would distort the Q R S group. A heart with the muscle in a poor physiological condition will tend to have small waves, no matter what the degree of hypertrophy, so that this factor will often reduce to normal or less the size of the excursions of the Q R S of a much hypertrophied heart. The effect of an abnormal path of the contraction wave due to myocardial disease is not predictable (Chap. IV).

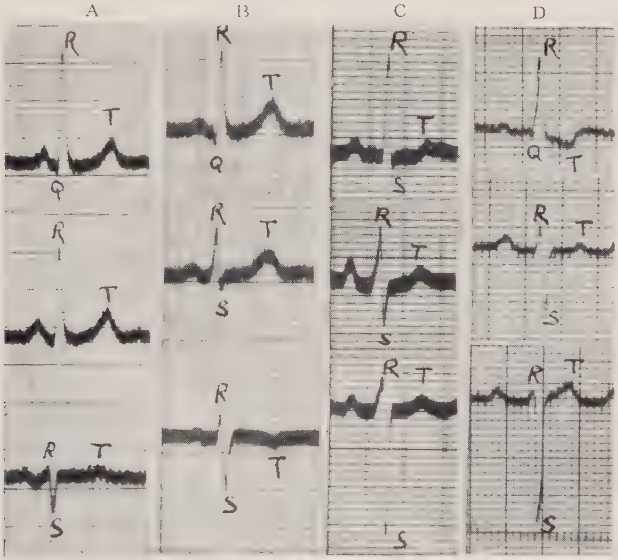

FIG. 7. Records from four different hearts to show different degrees of left axis deviation of Q R S. Record A has the least marked deviation and Record D the most. Note that S 3 increases in size in relation to R 1 as the degree of deviation increases, until in Record D, S 3 is larger than R 1. In Records C and D, S 2 shows an increasingly large deflection. In Record D, Q R S has a duration of .12 sec.

Increased Duration of the Q R S Group. In many records with marked left axis deviation the duration of Q R S is increased to more than .10 sec. The prolongation of Q R S in such records is due to the increased thickness of the ventricular muscle through which the contraction wave must pass in going from the endocardial to the pericardial surface. We know from experiments upon dogs that the last part of the ventricular muscle to become involved in the contraction is at the basal part of the left ventricle. Since Q R S lasts until the whole of the ventricular muscle has entered into contraction, it is readily understandable why thickening of the left ventricular muscle causes a prolongation of Q R S. The rate at which the contraction passes through the ventricular wall of the dog is 5 mm. per .01 sec. If it were the same rate in man then an added centimeter of thickness of the ventricular wall would prolong Q R S for .02 sec.

We should not, then, consider a duration of .11 sec. abnormal for the Q R S of records showing marked left axis deviation and if the heart is very large and the axis deviation very marked, as in Figure 7 D, even .12 sec. may be considered due to the hypertrophied ventricular wall and not to disease. Records showing marked right axis deviation do not show this prolongation of Q R S, because the wall of the right ventricle is not the latest region to enter into contraction. Any increase in the time of spreading through the right ventricular wall is included within the normal duration of Q R S.

The T Wave. The T wave is not changed by ventricular hypertrophy as is the Q R S group. Records with marked right axis deviation (Fig. 6 D) or marked left axis deviation (Fig. 7 D) have a T wave in no way different from normal. This is an interesting fact which has not been given sufficient emphasis by those concerned with the theory of the cause of T.

In a large series of records in which I have studied this feature, it was found that T 2 and T 3 were directed downward in about 25 per cent of the records showing right axis deviation while T 1 was directed downward with or without T 2 in about the same percentage of records showing left axis deviation. On the other hand, T 2 and T 3 were very rarely turned downward in records with deviation to the left (3 per cent) and T 1 was as rarely downward in records with deviation to the right.

It is plain that the direction of the deviation of the axis is somewhat concerned in the direction of T in the different leads, but it is not a controlling factor, else T would be affected in accord with the axis deviation in much more than 25 per cent. Moreover, a marked degree of axis deviation may be recorded and the T wave be upward in all three leads. It seems as though the axis deviation of Q R S were a directing factor, so that when the contraction is affected by some other influence, T 1, and perhaps also T 2, become inverted in the presence of left axis deviation, while with right axis deviation, T 2 and T 3 are inverted.

Effect of Position of Heart

It will be well, here, to discuss further the effect upon the electrocardiogram of the position of the heart in the chest. We have pointed out that this can modify the record, varying the relative size and direction of the waves in the three leads. It acts in this way upon all the waves, but most upon the Q R S group, and least upon P. The influence upon the electrocardiogram of the changed position of the diaphragm during expiration can be well seen in Figure 8 where Lead 3 shows a gradual decrease in the size of R with coincident increase in the size of S. If the other leads of this record were examined it would be seen that in them also there was a slow waxing and waning of the height of the waves.

These variations are due to the movement of the diaphragm, making the heart lie more transversely at full expiration and more vertically within the chest when the diaphragm sinks with inspiration. The heart is relatively fixed at its base by the attachments of the arteries and veins, and the apex is relatively movable, so that the rise and fall of the diaphragm rotates the longitudinal diameter of the heart upon the base as a fixed point. It will move, as we view the patient from in front, in a counter-clockwise direction during expiration and in a clockwise direction during inspiration.

This results in a change in the electrical axis of Q R S and of T. P is changed to a lesser extent because the auricular part of the heart is less freely movable.

A fluoroscopic examination may reveal the extent of this rotation to be as much as 30° in some hearts. The electrocardiogram by the three leads *must* vary if the heart moves as much as this, for if anything but a circle should rotate as much as 30° it would be sure to have a different appearance when viewed from a constant direction, such as is represented by each of the leads (Chap. IX).

Few hearts, however, vary as much as 30° with respiration, the usual rotation being about 15°. Often the diaphragmatic excursion is slight, and often the heart, especially if of the vertical type, will rest so lightly upon the diaphragm as to be scarcely affected by its motion. Respiratory variation of a wave appears most plainly in whichever lead gives it the smallest relative value, for in this lead the rotation of the heart can change the wave from an upward to a downward variation or vice versa. This is why the vibratory type of Q R S is especially likely to show plain variations with the respiratory movement.

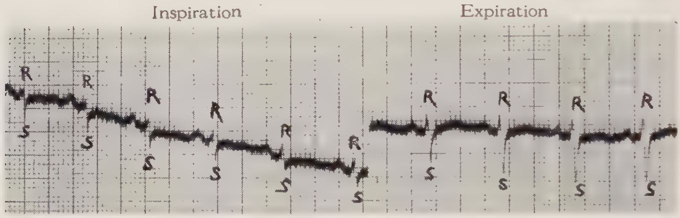

Fig. 8. Electrocardiogram by Lead 3 showing the change in Q R S during forced expiration. At the left of the figure during full inspiration the R and S waves are about equal. At the right of the figure during full expiration the S wave has become much larger.

If the diaphragm is permanently high in the thorax, a condition usually associated with a broad hypersthenic type of chest (Fig. 9 A) or with an obese abdomen, the heart will be permanently rotated so that its long axis will lie horizontally. The effect on the waves of the electrocardiogram will be to change their proportional value in the three leads, so that P and T are likely to be relatively small in Lead 3, or T may even be inverted. The Q R S group is likely to resemble those of Records E, F, G and H of Figure 5, or even Record A of Figure 7, the latter indicating a slight left axis deviation of the Q R S group. If a heart which had an L/R ratio with the balance slightly too far toward the right should be associated with a high diaphragm, this slight *preponderance* of the right ventricle might be concealed and the Q R S be normal. Likewise, a normally balanced heart with a high diaphragm would tend to show a Q R S with slight left axis deviation, and one with an L/R ratio slightly toward the left yet within normal limits, would be certain to show a definite left axis deviation of Q R S.

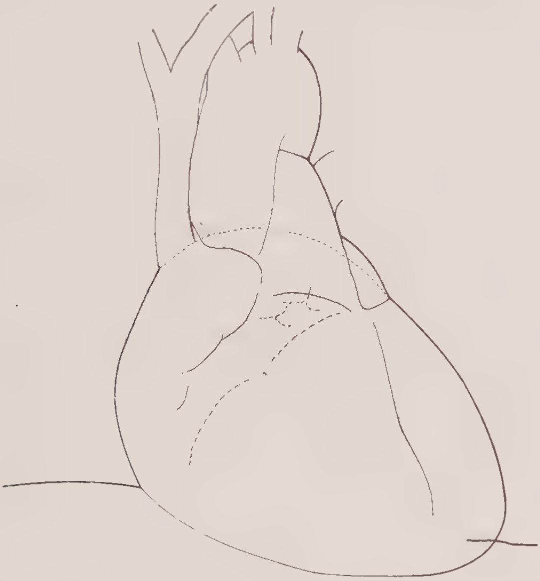

FIG. 9A. Silhouette area of the heart and great vessels of a stocky man. Inside the silhouette area the ventral surface of the heart and the position of the valves have been schematically represented. After Bardeen (*Am. J. Roentgenol.*, Dec., 1922, ix, 827).

A permanently low diaphragm such as is usually associated with a long narrow chest has just the reverse effect on the waves of the record. The heart hangs more vertically, and the effect on the record is that P and T are likely to be relatively small in Lead I, and Q R S to have characteristics like those of Records A and B of Figure 5. T will never be turned downward in Lead I by this position of the heart as it may be in Lead 3 by a transverse position. In the long narrow thorax a heart with an L/R ratio with the balance toward the right, yet within normal limits, might give an electrocardiogram like Figure 6 A, showing slight right axis deviation of Q R S. One with an L/R ratio with the balance too far toward the left, just beyond the normal, might give a record of the type of C or D of Figure 5, not indicating the left *preponderance* which was present.

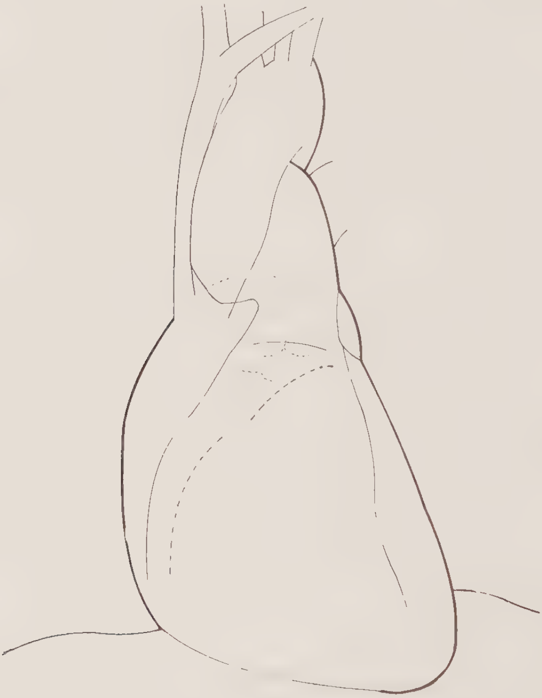

FIG. 9B. Silhouette area of the heart of a relatively slender man. After Bardeen (*Am. J. Roentgenol.*, Dec., 1922, ix, 828).

Some very important work has recently been done which goes to show that the rotation of the heart upon its long axis has a similar effect upon the Q R S group to that which has been described for variable positions of the diaphragm. Rotation of the anterior surface of the heart to the right tends to produce right axis deviation of Q R S and rotation to the left, left axis deviation. It is very possible that the movements of the heart upon the diaphragm with respiration or with forced breathing are accompanied by a rotation about the longitudinal axis and that it is for this reason, as well as because of the actual change in the anatomical axis of the heart, that the Q R S group varies as it does.

When either ventricle is more than slightly hypertrophied so that the relation of its weight to the other is decidedly abnormal, the influence of this *preponderance* upon the Q R S group is strong enough to overbalance the effect of an abnormal position of the heart or an unusual distribution of the bundle-branch tissue within the ventricles. Slight degrees of mass preponderance may only serve to balance the opposite type of electrocardiographic signs which may have resulted from an abnormal position of the heart or from an unusual distribution of bundle-branch tissue. For example, a vertical heart whose normal electrocardiogram is like that of Figure 5 A would need more mass preponderance of the left ventricle to produce a left axis deviation of Q R S than would a transverse heart whose normal record was like that of Figure 5 F. With more markedly preponderant hypertrophy of either ventricle, the features of position and bundle-branch distribution become of diminishing importance. To state this in another way: an enlarged heart which gives a record of either right or left axis deviation is much more likely to have this because of a muscle preponderance than is a heart with the same degree of electrocardiographic variation, but without enlargement.

The effect of lateral displacement of the heart upon the electrocardiogram is often quite characteristic, so that it may be recognized from the record. This is especially true if the displacement is toward the right side. The change consists in a deepening of Q and S in two or more leads, or their appearance along with a diminution in the height of the R wave. Each limb of the R wave thus appears to be

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suspended below the zero level of the record. This curve is found almost without exception when the heart is pushed to the right by pleural fluid or air, or pulled over by pleural adhesions or fibrosis of the lung, and gives a very different picture from that due to congenital dextrocardia. Displacement to the left is less liable to change the electrocardiogram in this way, and in fact usually changes it very little.

Clinical Application of the Diagnosis of Axis Deviation

Remember that right or left axis deviation does not exclude hypertrophy of the other ventricle. It is common to find a greatly hypertrophied heart whose electrocardiogram has a Q R S group, with a normal relation of the size of the waves in the three leads. This indicates very clearly that the two ventricles have become hypertrophied and that the left has hypertrophied more than the right so that the normal relation of their masses is retained. In considering the significance of right or left axis deviation of Q R S, both the actual size of the heart and its position must be included in the picture. Having in mind these three factors, the position, the size and the direction of the axis of Q R S, we can appraise the size of each ventricle separately. Should there be evidence of hypertrophy of either chamber, the diagnosis is not completed until we have discovered the cause of this hypertrophy.

Auricular hypertrophy will appear from the changes in P as described. It may be the result of venous congestion in either the pulmonary or the systemic veins. The former is probably far more common and the accepted electrocardiographic signs of auricular hypertrophy probably depend mostly upon a hypertrophied left auricle. Mitral stenosis causes the most marked auricular hypertrophy, but mitral regurgitation has a similar though less marked effect. Disease of the aortic valve will increase the work of the auricles when the left ventricle fails to empty itself properly, and the same is true of high arterial tension; thus these conditions lead only secondarily to auricular hypertrophy.

Hypertrophy of the right ventricle may be caused by disease of the pulmonary arteries with narrowing, usually a syphilitic condition. Chronic emphysema and chronic pulmonary tuberculosis often lead to hypertrophy of the right ventricle, the narrowed pulmonary capillary bed probably being the cause. Certain congenital abnormalities of the heart cause great hypertrophy of the right ventricle. Pulmonary stenosis, patent interventricular septum and patent ductus arteriosus obviously would do this, but a patent foramen ovale or a prominent ventricular band would not. Congenital cardiac defects may also be accompanied by abnormalities of the branches of the auriculoventricular bundle, so that at times the hypertrophy will be accompanied by an abnormal path of the contraction wave. This may even mask the electrocardiographic sign of hypertrophy of the ventricle.

Mitral stenosis leads to an hypertrophy of the right ventricle through a damming back of blood in the pulmonary capillaries, thus increasing its work. Mitral regurgitation does this also, though secondarily and only after overtaxing the left ventricle. Hence the earliest records with this valve lesion may show a left axis deviation of Q R S, later ones giving a normal Q R S, and later still, as stenosis develops, a right axis deviation.

Hypertrophy of the left ventricle is caused by disease of the aortic valve, either regurgitation or stenosis, by arterial hypertension, by arteriosclerosis, by mitral regurgitation and by diffuse myocardial degeneration. Long-continued strenuous physical exertion may lead to left ventricular hypertrophy, though more often to an hypertrophy of both ventricles so that Q R S is not changed. Arterial hypertension and aortic stenosis cause the most marked left ventricular hypertrophy; and as they affect the right ventricle only secondarily through failure of the left, they lead to marked grades of left axis deviation of Q R S.

Though the determination of a *preponderant* hypertrophy of either ventricle is not the most important information derived from the electrical curves, yet it cannot be obtained in any other way, and often throws an important light upon a doubtful diagnosis. The presence or absence of hypertrophy of the right ventricle is often a factor in deciding whether the rumbling diastolic murmurs heard at the apex when aortic regurgitation is also present are due to a coincident mitral stenosis or to the aortic regurgitation by the mechanism described by Flint. The presence of a right axis deviation is a proof that mitral stenosis is present, though it is not uncommon to find a Q R S with the normal relation of the waves in the three leads, due to a balanced hypertrophy from a slight degree of stenosis combined with the aortic lesion.

Two valvular lesions deserve special mention. A pure uncomplicated mitral regurgitation will usually give an electrocardiogram showing a slight or moderate degree of left axis deviation, or a normal ventricular relation, irrespective of the amount of hypertrophy of the heart. Uncomplicated aortic regurgitation is usually associated with left axis deviation, but there are a certain number of patients who fail to show this. Most, if not all of these, have little or no enlargement of the heart, and are often without the increased pulse pressure so characteristic of this valve lesion. Yet the murmur may be quite typical and often fairly loud. Some of these patients have a vertical position of the heart which could mask a slight left-side hypertrophy as previously mentioned, but in other cases it is impossible to explain the lack of abnormal axis deviation.

The clinician is sure to wonder *how exact a measurement of relative ventricular size this electrocardiographic sign has been found to be.* It is in general correct, and especially if there are evidences of cardiac enlargement. For final decision the matter needs further investigation, but the clinical pathological studies so far reported correlating the direction of the electrical axis of Q R S with the relation of the ventricular weights, show that, in 75 per cent of instances, the electrocardiogram will suffice to place the ventricular relations, and that the error usually results from the record indicating slight right or left axis deviation in cases with normal weight relations. Those hearts with a definite pathological *preponderance* of either ventricle give a definite axis deviation of the Q R S group. The discrepancies between the records and the ventricular weight ratios are usually of such a character that they might well have been due to a modification of the Q R S group by a vertical or a transverse position of the heart within the chest. At times an extreme variation of the electrical axis may be found without cardiac enlargement. This is always difficult to explain but sometimes may be due to pathologic changes in the muscle.

How shall we best summarize the Q R S group so as to use it to place the records as correctly as possible in the scale of ventricular preponderance? This question must await its final answering, but there are two general types of procedure, either of which will serve fairly well:

Einthoven determined the direction within the heart of the potential which produced the largest excursion of the Q R S group (see appendix). He considered that those hearts without hypertrophy of either ventricle would have the largest potential of Q R S within the sector between 40° and 90° below a line drawn horizontally to the patient's left. Those which had this potential directed above 40° showed left ventricular hypertrophy and those which gave a direction nearer the patient's right than the vertical, which is 90° below horizontal, were hearts showing right ventricular hypertrophy.

Lewis suggested a method of obtaining an index of ventricular preponderance by the formula (R 1 − R 3) + (S 3 − S 1), the height in millimeters of the waves in the leads being indicated by R 1, S 1, R 3 and S 3. He did not mention what he considered to be the normal limits for the result of this formula, expecting probably to determine this by a sufficient number of autopsies with weighing of the separated ventricles. White suggested a very similar formula (U 1 + D 3) − (D 1 + U 3) in which U is the height in millimeters of the largest upward deflection of Q R S and D is the height of the largest downward deflection in the leads indicated by the figures. The result of this formula will differ from that of Lewis' formula only when Q is larger than S in either Lead 1 or Lead 3.

Lewis' formula is more sound theoretically than White's, for the Q and the S waves of Leads 1 and 3 are apparently governed by different ventricles, and not by the same ventricle, as they are considered to be in White's formula. In an attempt to determine what the normal limits might be for Lewis' formula, the author reviewed 30 cases, published in part by Lewis and in part by Cotton. The ventricular weights and electrocardiographic records of these patients were compared, and for hearts without preponderant hypertrophy of either ventricle, the index figure usually lay between +20 and −10. A figure greater than +20, then, should indicate left ventricular *preponderance*, and one less than −10, right ventricular *preponderance.*

Lewis' formula has two great faults: (1) We do not know how to correct it for rotation of the long axis of the heart, as we do the angle of Einthoven's method; and (2) it is often dependent upon the actual amount of potential produced in the heart, and not upon its upward or downward direction in the three leads, for it is plain that a value of 20 could not possibly be obtained from a record whose largest excursion during Q R S was but 6 mm., e.g., R 1 = 4 mm., S 1 = 0, R 3 = 1 mm., S 3 = 6 mm. Lewis' index is 9 [(4 − 1) + (6 − 0)] for this record with very marked left axis deviation of the Q R S group.

Einthoven's method is not difficult to apply, though it involves a little mathematics. In records in which the highest peaks of the Q R S groups occur at the same instant in the three leads, so that the height of Lead 1 + that of Lead 3 = that of Lead 2, it is easy to apply Einthoven's table to determine the direction of the current within the heart (see appendix); but in records with the high peaks "out of phase" so that the above formula is not correct for the height of the peaks, it may not be possible to determine *exactly* the direction of the angle of the largest excursion without a laborious magnification and measuring of the waves. It can, however, be obtained approximately, which is enough for clinical purposes.

When the two largest waves of Q R S occur in Leads 1 and 2 or in Leads 2 and 3 it is usual to find these two peaks representing the same instant of the Q R S group—"in phase,"—and their values should be used to derive the values of the other lead. For example: Figure 6 A gives R 3 = 13 mm., R 2 = 11 mm. Hence,

$$QRS\,1 = R\,2 - R\,3(11 - 13) = -2 \text{ mm.}$$

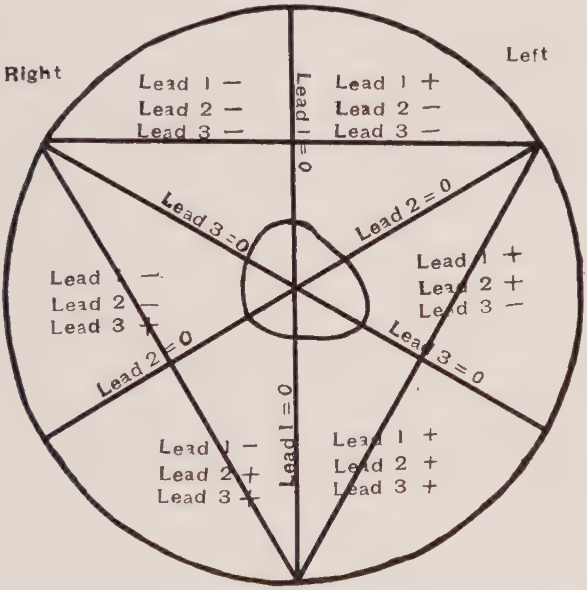

FIG. 10. A schema to present graphically the direction of the deflections in the three leads resulting from a current within the heart with any direction in this plane. The circle is divided into six sectors; when the heart's current is parallel to any of the radii within a sector, the deflections in the three leads will be upward (+) or downward (−) as shown. When the heart's current is parallel to the side of a sector, one of the leads will have no deflection from this current, but the other two will have equal deflections.

Figure 7 A gives R 1 = 15 mm., R 2 = 12 mm. Hence,

$$QRS\,3 = R\,2 - R\,1(12 - 15) = -3 \text{ mm.}$$

When the two largest waves are found in Leads 1 and 3 it will not be possible to predict which peaks will fall at the same time instant. We must note this by inspection of the record measuring from the beginning of Q R S of each lead to the respective peaks. When the axis deviation has reached such a marked degree as this the exactness of the height of the waves is of relatively less importance, for slight differences in the height make negligible differences in the angle.

Neither the Lewis nor the Einthoven method of measuring ventricular *preponderance* is more than approximate because of the variations due to position of the heart and to varying distribution of the bundle arborizations. On this account, it has seemed to the author that for clinical purposes it will suffice to view the Q R S group pictorially, as it were, and to determine the direction of the axis of the largest deflection of Q R S by noting the direction of the largest excursion in each lead. When the values in the three leads do not approximately fulfil Einthoven's law of 1 + 3 = 2, it is better to compute that value which is the smallest from the two larger by substituting them in the above formula. Frequently, by using the vertical time lines carefully, measuring from the beginning of the Q R S in each lead, we can arrive at a very good idea of coincident time points in the three leads.

When the values are all upward (+) the leads may be represented by + + + and the angle of this potential lies within the sector of normal balance (+ 30° to + 90°) as may be seen by referring to Figure 10; if Lead 1 or Lead 3 is small in relation to the others, then the angle of the potential lies near the border of that sector, which if entered would give a negative (−) value for this lead and would constitute a right or left deviation of the axis of Q R S. We must now consider the long axis of the heart, noting whether it is unusually vertical or transverse. If so we must correct the angle of the chief potential of Q R S for this abnormal rotation of the heart, turning it a little in the direction of the movements of the hands of a clock if the heart lies too horizontally and counter-clockwise if the heart is too vertical.

A moderate right or left axis deviation will be shown by the values in the leads being − + + or + + − respectively, with the negative value always less than the value of Lead 2: e.g.,

Lead 1 = − 2, Lead 2 = + 8, Lead 3 = + 10,

or

Lead 1 = + 10, Lead 2 = + 6, Lead 3 = − 4 as in Figures 6 A and 7 A. If the value in Lead 2 is less than the value in Lead 1 in records with right axis deviation, e.g., − 6 + 4 + 10, then we may consider it more than moderate in degree, as in Figure 6 B. If Lead 2 becomes zero or less (Fig. 6 C and D) then the degree may be considered as a marked one. In records with left axis deviation, the condition is analogous, values of + 10 + 4 − 6 being a moderate degree (Fig. 7 B and C) and + 8 − 2 − 10 a marked degree of left axis deviation (Fig. 7 D).

Examining the relative values of the three leads in the portion of Einthoven's first table (see appendix) which lies between − 50° and + 160° will give a good idea of the reciprocal variations of these values. In practice they may be fitted pictorially into the scheme of Figure 10.

When following records of an individual case over a period of time it is desirable to be more exact than this, but for general clinical diagnosis such a grouping as has been outlined will be found quite exact enough.

SUMMARY

The electrocardiographic diagnosis of auricular hypertrophy should probably be stated as hypertrophy of the left auricle. This diagnosis may be made when the P wave is found to be over 2 mm. in any lead, to be notched and to have a duration of .12 seconds, or if two of these peculiarities are found.

In the diagnosis of ventricular hypertrophy we are able to determine with fair accuracy whether the right or the left ventricle is chiefly affected. The greater the cardiac enlargement, the greater our accuracy in this respect. In making this diagnosis we must consider the direction of the electrical axis of Q R S and the direction of the anatomical axis of the heart as determined by a vertical or a transverse position of the heart within the chest.

Hypertrophy of the right ventricle may be diagnosed with an enlarged heart if right axis deviation of Q R S is found. Without cardiac enlargement it should not be diagnosed if the heart is of the vertical type with a low diaphragm, unless right axis deviation is marked; with a normal or transverse position of the heart and right axis deviation hypertrophy of the right ventricle is probable.

Hypertrophy of the left ventricle may be diagnosed with an enlarged heart if left axis deviation of the Q R S is found. Without cardiac enlargement it should not be diagnosed if the heart is of the transverse type with high diaphragm unless left axis deviation is marked; with a normal or vertical position of the heart and left axis deviation hypertrophy of the left ventricle is probable.

Hypertrophy of both ventricles may be diagnosed with an enlarged heart if there is neither right nor left axis deviation of Q R S.

Chapter IV

CHANGES DUE TO MYOCARDIAL DISEASES

The most important feature of electrocardiographic diagnosis depends upon the fact that myocardial disease can change the electrical production of the heart beat, and thereby produce abnormal waves in the electrocardiogram. Such electrocardiographic abnormalities will be present whether the heart is regular or irregular, fast or slow. They depend upon the abnormality of the muscle of the chambers concerned, and therefore are found each time the chambers contract.

Abnormal types of contraction, such as premature beats and tachycardias, can also produce abnormal waves in the electrocardiogram. These waves are readily distinguished from those due to disease of the muscle, by their relatively infrequent occurrence, or by the fact that they are present in one record and absent in another. Such transient variations are associated with an abnormal cardiac rhythm.

Functional variations in the muscle can also cause changes in the electrical waves. The electrical production will be changed if the muscle is below par because of fatigue or a failing blood supply, or because of the action of drugs or toxins. These are temporary variations, lasting only as long as the causative agent is active. A single record may not suffice to tell whether an abnormality is due to abnormal function or to disease; but a later record or a review of the clinical features of the case should enable us to decide.

Abnormality of the P Wave

Auricular fibrillation or auricular flutter may usually be taken as evidence that the auricular muscle is diseased (Chap. VII). Premature auricular beats or auricular tachycardia often result from disease also, but this is not always the case. Except for these conditions we learn but little about the auricular muscle from the changes in form shown by the P wave. There has not yet been enough study of autopsy material to justify more than a surmise as to the cause of P wave abnormality.

The P wave is sometimes very abnormal in appearance, as in Figure 11 A and B, and such abnormal waves persist.

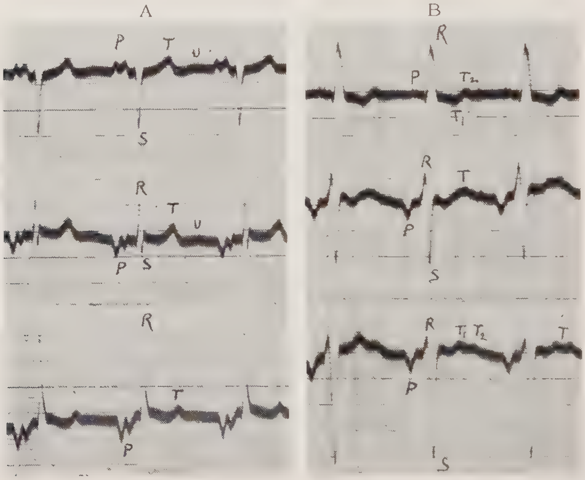

Fig. 11 A. Abnormal P waves, notched and with increased duration. The ventricular complexes show right axis deviation of Q R S.
B. Abnormal P waves, inverted in Leads 2 and 3. The ventricular complexes show marked left axis deviation of Q R S, a curious double-topped T wave in Lead 3 and a diphasic T wave in Lead 1.

We believe this abnormality is due to a focal disease in the auricular muscle, yet we do not *know* that we are correct. Inversion of P in Leads 2 and 3, or in Lead 1, with or without notching of the wave, or even the occurrence of marked notching alone, are changes which would be expected to result from the abnormal contraction due to disease of the auricular muscle. No correlation of pathological and electrocardiographic data has been reported on this subject, however, and the only experimental work which has come to my attention bearing upon it is a single record published by Lewis to illustrate another matter, in which marked notching and large size of P were present after one auricular appendix had been crushed. Notching of P is the sort of change which we would expect from an injury of this sort, and disease might in a similar way lead to an interruption of the normal path of the contraction wave, or to an uneven spreading of the contraction to the right and left auricles.

Unless it leads to arrhythmia, auricular myocarditis has little effect upon the functional ability of the heart, for the force of the heart-beat is in the ventricular contraction. Myocardial disease is but rarely localized in the auricles, however, so that an ability to diagnose auricular myocarditis will often give a better insight into the pathological processes of certain patients in whom ventricular disease is either absent, or is insufficient to change the ventricular curves.

When P has no larger excursion than 1 mm. in any lead, we have an indication of a poor state of nutrition, a poor functional condition of the muscle. Only 7 cases (10 per cent) in the combined normal series of 78 cases of Lewis and the author gave a figure of less than 1 mm. for the P wave, and only 1 of these less than 0.7 mm. An abnormally small P wave might also result from a diffuse disease of the auricular muscle. A focal process would be more likely to cause a notched P wave. Small P waves are seen after prolonged infectious diseases, and in hearts which are considered to have narrowing of the coronary arteries. In both instances the factor of poorly nourished muscle is evidently present. The P wave has often been seen to increase in size during convalescence from infectious diseases and after recovery from cardiac failure, and as both are occasions when the condition of the muscle is improving, these observations are cited in further support of the hypothesis.

Since a hypertrophied auricle will give a P wave of greater than normal size when its muscle is in good condition, it will be rare for the P wave from such a heart to sink below the size which has been considered a minimum for auricles which are not hypertrophied.

Abnormal Ventricular Waves

Each of the waves of the ventricular complex is subject to variation in height, width and form as a result of disease of the ventricular muscle. The resulting ventricular complexes, therefore, differ extremely from one case to another, and it is rare for the complexes from different patients to have more than the most general resemblance to each other. The Q R S group may be large or small, increased in width, or notched, and along with these changes the T wave may be large or small, or may be inverted in one or more leads. Certain changes tend to occur together in special combinations, but it is after all a rather uncommon occurrence, so that we find very many forms of abnormal ventricular complexes.

Bundle-branch Lesions. The combination of electrocardiographic abnormalities which occurs most frequently is associated with quite definite pathological changes. It will serve as a sort of introduction to the discussion of the changes in the ventricular complex due to disease.

If, in a dog, pressure is made over the branch of the auriculoventricular bundle which carries the stimulus from the main bundle to the right ventricle, the passage of this stimulus will be blocked. The left ventricle then, is the only one to be affected by the impulse from the A-V node. The left ventricle is the first to contract, and the right ventricle becomes involved only by the spreading of the contraction from the left ventricle through the interventricular septum to it. With this change in ventricular contraction the form of the ventricular complex immediately changes to one similar to those of Figure 12 A or B, which are from human hearts.

The typical characteristics of these records are: (1) The ventricular complex has an abnormally wide Q R S group, the duration being for the human heart at least .14 sec., and usually more than this. (2) The largest wave of Q R S is oppositely directed in Leads 1 and 3, either upward and downward as in Figure 12 A, or the reverse of this, if the left bundle branch is affected, as in Figure 13 A. (3) The Q R S group always shows notching or thickening of the ascending or descending portion in more than one of the leads, or there may be a notching of one of the peaks. (4) The T wave is opposite in direction to the largest wave of Q R S in all three leads, but exceptions to this rule occur when the Q R S group in one lead has a small relative value, or has both upward and downward waves, as in Figure 12 B. Under either of these circumstances the T wave in this lead may be very small in size, or directed both upward and downward (diphasic), or directed opposite to the last deflection of the Q R S group. (5) The size of the largest deflection of Q R S in its largest lead, is often well above the average normal figure of 12 mm. (6) The height of the T wave in its largest lead is often greater than the maximum 5 mm., which is normal for T. (7) If the P wave is present, it is followed by the abnormal ventricular complex after a P-R interval

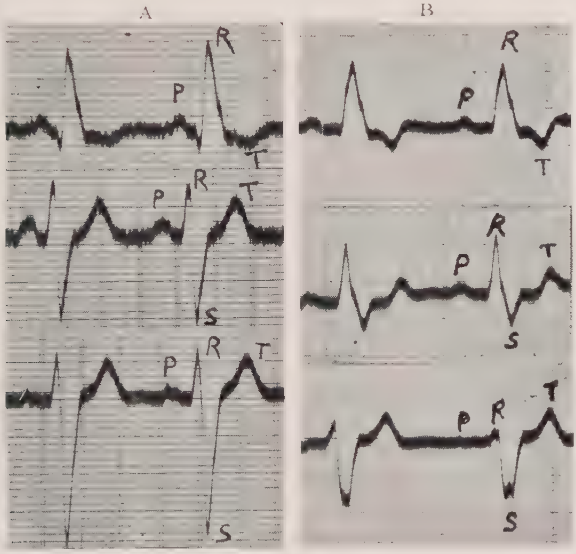

Fig. 12. A and B indicate that the right branch of the auriculoventricular bundle is not functioning—right bundle-branch block. Note the abnormal width of the Q R S group, the notching of Q R S, and the abnormal direction of T in Lead 1. The normal P-R interval of these records shows that the function of the main bundle is normal.

which is constant from beat to beat and is usually normal; or if auricular fibrillation should be present, these abnormal complexes will occur with the irregularity characteristic of this condition, as in Figures 13 B and 15 A.

The increase in the duration of Q R S is due to the contraction taking an abnormally long time to involve the whole of the ventricular mass. This it does because it must now spread from one ventricle to the other through the ventricular muscle, instead of being distributed to both ventricles almost simultaneously by the right and left branches of the bundle of His and their ramifications. When the contraction has spread through the septum from one ventricle to the other it is distributed throughout the second ventricle by means of the branches of the auriculoventricular conducting tissue within that ventricle. That is, it passes from the muscle of the septum to the ramifications of the conducting tissue and thence through this tissue to the remainder of the muscle of the other ventricle. The contraction passes through the

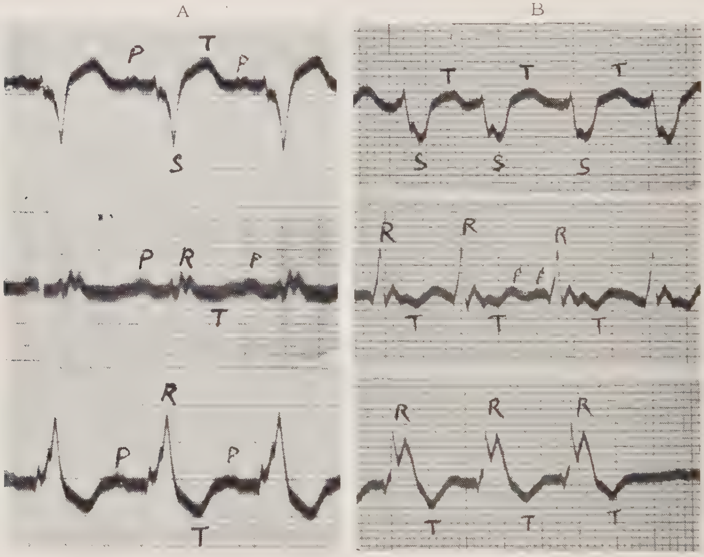

Fig. 13. Abnormal ventricular complexes indicating left bundle-branch block. The abnormal width and notching of the Q R S group and the downward T waves in Leads 2 and 3 are the important features. Record A has normal auriculoventricular conduction time. Record B shows auricular fibrillation.

ventricular muscle at a much slower speed than along the auriculoventricular conduction system. In the dog's heart the figures are 500 mm. per sec. for ventricular muscle versus 5000 mm. per sec. for the conducting tissues. During the normal ventricular contraction the distribution of the impulse to the whole of the inner surface of both ventricles is probably completed in .02 or .03 sec. The remainder of the .08 or .10 sec. needed for the complete spreading of the contraction (shown by the duration of the Q R S group) is occupied in its passage through the thickness of the ventricular wall. It is plain from this why the spreading of the contraction through the septum would greatly prolong Q R S.

The large size of these abnormal Q R S groups and T waves is due to the fact that the normal ventricular waves result from a balance of potentials coming from the right and left ventricles simultaneously, which are for the most part in opposite directions. When there is an interruption of the function of one bundle branch the contraction involves one ventricle an appreciable time before it reaches the other. This results in a lack of the usual partial electrical balance at the beginning and end of the contraction, and hence the large waves.

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We can determine, from the direction of the chief deflections of Q R S in the three leads, which ventricle is the first to enter into contraction. From this we deduce that the bundle branch to the opposite ventricle is affected. The sign of right- or left-side precedence is similar to that which indicates *preponderant hypertrophy* of the corresponding ventricle. If the chief deflection of Q R S is downward in Lead 1 and upward in Lead 3, as in Figures 13 A and B, then the right ventricle precedes, and the lesion is of the left bundle branch. If the chief deflection is upward in Lead 1 and downward in Lead 3, as in Figures 12 A and B, then the left ventricle precedes, and the lesion is in the right bundle branch. When the right ventricle precedes the left the Q R S group shows right axis deviation, and vice versa.

These abnormal ventricular complexes bear a resemblance to those due to premature beats arising in one or the other

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ventricle, as can be seen by comparing Figures 12 and 13 with Figures 31 and 32. The ventricular precedence is indicated in the same way in each case. It apparently makes little difference to the electrical curve whether a contraction starts in one ventricle spontaneously or is started there first by a stimulus along the normal paths. The chief difference between the complexes due to bundle-branch lesion and those due to premature ventricular beats is that the latter are apt to have larger waves, and tend more frequently to have a sharp notching of the peak of the large Q R S waves.

The complexes which result from a stimulus coming to the ventricles along only one of the branches of the auriculoventricular bundle have been named by Lewis levocardiograms or dextrocardiograms, depending upon whether the left or the right ventricle first receives the stimulus. In the one case the form approximates Figure 12 A and B and in the other, Figure 13 A and B. The normal electrocardiogram obtained when both bundle branches are functioning normally he calls a bicardiogram. He shows that from the levocardiogram and dextrocardiogram of the same heart the Q R S group of the bicardiogram may be reconstructed by a process of summation of the electrical potentials recorded at corresponding instants, throughout the early stages of the spreading of the contraction in the separate ventricles.

Besides these typical complexes of Figures 12 and 13, others less typical are found, such as Records A and B of Figure 14, and A of Figure 15. These may fail to have Q R S predominantly opposite in Leads 1 and 3, making it difficult to say which of the ventricles is first stimulated to contraction and just where in the Purkinje system the lesion may be. We believe, however, from the width and the notching of the Q R S group, and the large T wave opposite in direction to the chief deflection of Q R S, that there must be a lesion of the conducting system responsible for such curves. Furthermore, because the auricular wave is present at a constant interval before each of the ventricular beats (with the exception of such a case as Figure 15 A, which shows the irregularity of auricular fibrillation) we know that these complexes are

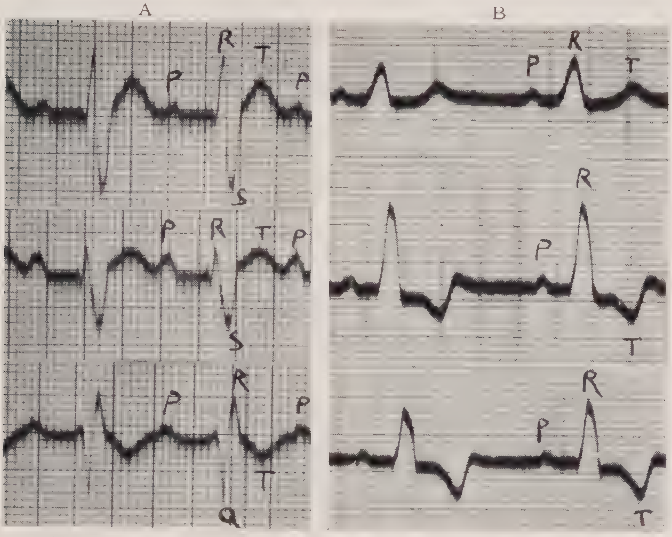

Fig. 14. Abnormal ventricular complexes indicating atypical bundle-branch block. The dysfunction is difficult to localize because of the atypical changes in the waves. Record A is probably due to a lesion of a part of the left branch and Record B to a lesion of parts of both branches. The auriculoventricular conduction is prolonged (.28 sec.) in Record A and is normal in B.

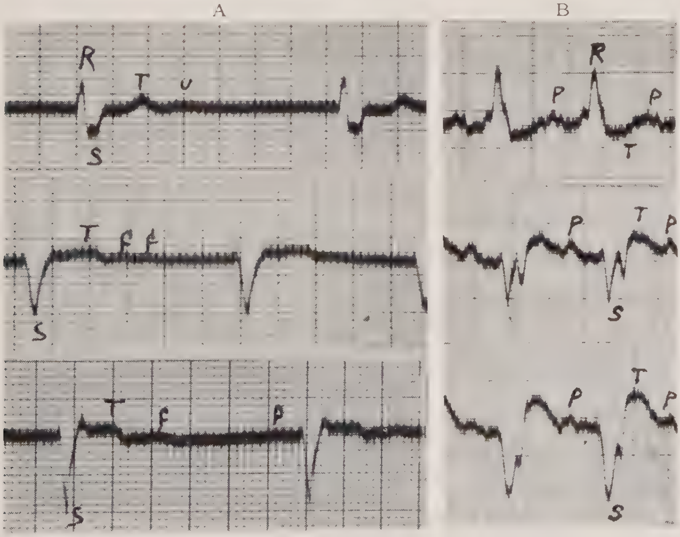

Fig. 15. Record A shows auricular fibrillation with ventricular complexes of atypical bundle-branch block, probably due to a lesion of a part of the left branch. Record B is a typical curve of right bundle-branch block with especially marked notching of Q R S. The auriculoventricular conduction time is .20 sec.

caused by supraventricular impulses coming along the A-V bundle from the auricles. When a supraventricular impulse results in ventricular complexes of this type, there must be an abnormality in the distribution of the impulse to the ventricles, to explain the abnormal form of the waves. Figure 14, A and B may result from a partial obstruction of the impulse in the left bundle branch, and Figure 15 A from a partial obstruction in both branches.

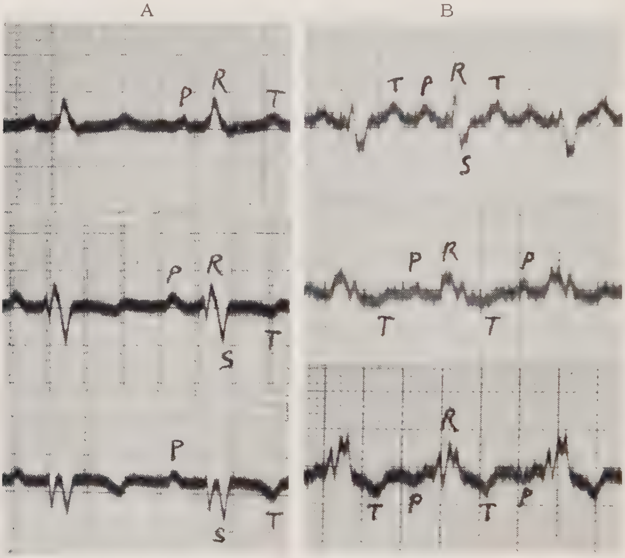

FIG. 16 A and B. Records showing abnormality of the spreading of the contraction with unusually small excursions of Q R S.
A. A later record of the patient of Figure 14 B showing changes in the complexes of the earlier record probably due to a change in the pathologic lesion.

Figure 16 A and B and Figure 17 A are examples of another variant of these curves, characterized by the small size of the excursions and the marked notching of Q R S. This type of complex has been ascribed by Oppenheimer and Rothschild to "arborization block" which they frequently found associated with a definite type of disease of the ventricular muscle, due in its turn to coronary arteriosclerosis. It seems likely from certain clinical reports that this type of ventricular curve does not always result from this sort of disease, and also that disease of a different sort can

lead to the production of this type of ventricular curve. The abnormal features of these curves, the notches and abnormal duration of Q R S and the abnormal direction of T indicate an abnormal ventricular muscle. The smallness of these deflections is, I believe, the result of a poor condition of the ventricular muscle. Figure 16 A is a later record of the patient from whom Figure 14 B was obtained, and shows a change in form suggesting the development of a lesion in the right bundle branch, the process in the left still remaining. Figure 17 A is a later record from the patient of Figure 13 A. The original form of the complex is, in the main, retained, the chief difference being in the size of the waves.

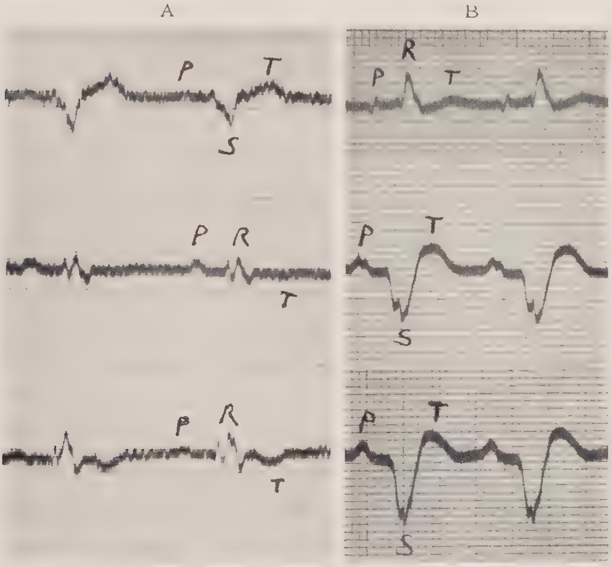

FIG. 17 A. A later record of the patient of Figure 13 A showing very little change in the complexes except diminution in size.

B. From a patient whose heart showed a sclerotic investment of the left bundle branch, the posterior half of this branch being replaced by connective tissue continuous with an adherent organized mural thrombus.

Notching of Q R S is an evident feature of these last records, and also of Figures 15 B and 17 B, which have larger waves. A marked notching of these complexes (more than the single notch commonly found at the peak of R or S) as in Figures 12 B, 13 B and 14 B, is probably due to secondary irregularities in the spreading of the contraction wave resulting from the areas of myocardial degeneration so common throughout these hearts (Chap. V).

The increased duration of Q R S is important in the diagnosis of bundle-branch defects, but it should be borne in mind that a marked hypertrophy of the left ventricle may also prolong the duration of this group. There may be a possibility of a difference of opinion as to the diagnosis of such records as Figures 11 B and 18 A, which have a Q R S duration of .12 sec. and which otherwise correspond in their characteristics with the curves of bundle-branch lesion, i.e., they have the Q R S group oppositely directed in Leads 1 and 3, and in these leads the T wave is directed opposite to the Q R S group. There would not, of course, be any question as to the presence of a bundle-branch lesion if the T waves were directed upward in all three leads as in Figure 18 B, for

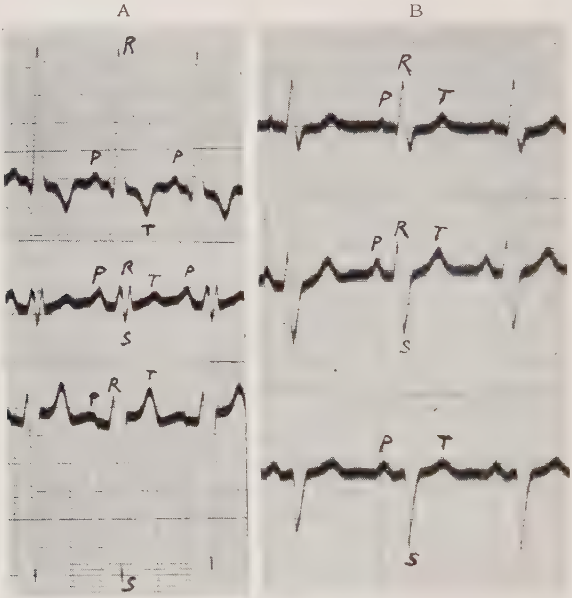

Fig. 18. Marked left axis deviation of Q R S with a duration of .12 sec. for Q R S. Neither of these records should be taken to indicate abnormal function of the right bundle branch, in spite of the fact that A has a T wave turned downward in Lead 1.

the record would be universally accepted as indicating no more than a marked left axis deviation of Q R S.

Such curves as those of Figures 11 B and 18 A are not uncommon, and I believe that when a record shows such a marked left axis deviation that Lead 2 gives a deflection approximately zero (Fig. 18 A) or definitely downward, (Figs. 11 B and 18 B) then a duration of .12 sec. for the Q R S group may be ascribed to the increased thickness of the ventricular muscle which must be traversed by the contraction (See Chap. III). This marked axis deviation must be due to left ventricular hypertrophy and the thickening of the muscle must be considerable. The contraction takes .01 sec. to pass through 5 mm. of ventricular muscle in the dog's heart, and possibly somewhat more in the human heart. Therefore an increase of .02 sec., (from .10 to .12 sec.) from this cause is readily understandable. A bundle-branch lesion produces a still greater widening of the base of Q R S, the figure reaching .14 sec. or more, as has been said.

Another conspicuous feature of these records is that their curves are very smooth and show an absence of the notching or slurring of the waves so characteristic of the bundle-branch lesion. The absence of notching seems additional evidence for the bundle branches being intact in these hearts, for it indicates a smooth and gradual development of the potential, a difficult thing to imagine if the contraction passed through the muscle of the septum from one ventricle to the other, as occurs with bundle-branch block. As a matter of fact, I have only very rarely seen a record with a duration of .12 sec. for Q R S without the characteristic notching being present, and then usually in the presence of marked cardiac enlargement with left ventricular hypertrophy.

Occasional records are found, which make it difficult to decide whether the abnormal width of Q R S, notching of Q R S and abnormal direction of the T wave indicate a lesion of the bundle-branch system. We shall be able to get at the basic condition in most records by remembering that though increased width of Q R S may be due to hypertrophy of the left ventricle, yet notching must be due to an abnormal spreading of the contraction. An abnormally inverted T wave may be due either to the latter or to a diffuse myocardial abnormality.

The diagnosis of right ventricular hypertrophy vs left bundle-branch lesion is less difficult, for the right ventricle does not increase in thickness to the same extent as the left. In fact, I have not seen a record with right axis deviation with a Q R S duration of .12 sec., except where notching was also present, indicating a possible lesion of the Purkinje fibers. Figure 19 A might perhaps be considered a record with a doubtful significance, for it has a Q R S duration of .12 sec., an atypical notched Q R S group of the right axis deviation type and a T wave which is always opposite in direction to the chief deflection of Q R S. The patient had a mitral stenosis of long standing and a much hypertrophied heart. This record was diagnosed to indicate a pathological lesion in the left bundle branch because of the long duration of the Q R S group combined with notching in the absence of marked left axis deviation of Q R S. The diagnosis was confirmed by the later development of complete obstruction of the left bundle branch during a period of acute cardiac failure, when Figure 19 B was obtained. This has a Q R S duration of .14 sec., and the Q R S is more typical of bundle branch lesion, in that it has no R wave in Lead 1 or S wave in Lead 3, that the T waves are of larger amplitude and that T is directed definitely downward in Lead 3.

A certain doubt has been cast upon the localization of the side of the heart in which these levocardiograms and dextrocardiograms arise. Figure 13 B is quite the type of record which is considered due to a lesion of the left bundle branch, and yet the heart from which it was made was reported by Oppenheimer and the author to show a destruction of the right branch of the bundle. Figure 15 B is of the other type, and yet the heart was found to have a lesion of the left branch with right branch quite intact. These pathological reports cannot be harmonized with the two reports by Eppinger and Stoerck showing a right-branch lesion with records like Figure 12 A and B. The situation suggests that there is some other unrecognized factor which varies the direction of the waves of these atypical complexes more than, or at least as much as, does the lesion of a bundle branch. We must await more facts before this question can be decided, but meanwhile we must hold to the localization which has been outlined, for all the experimental and theoretical considerations are in its favor.

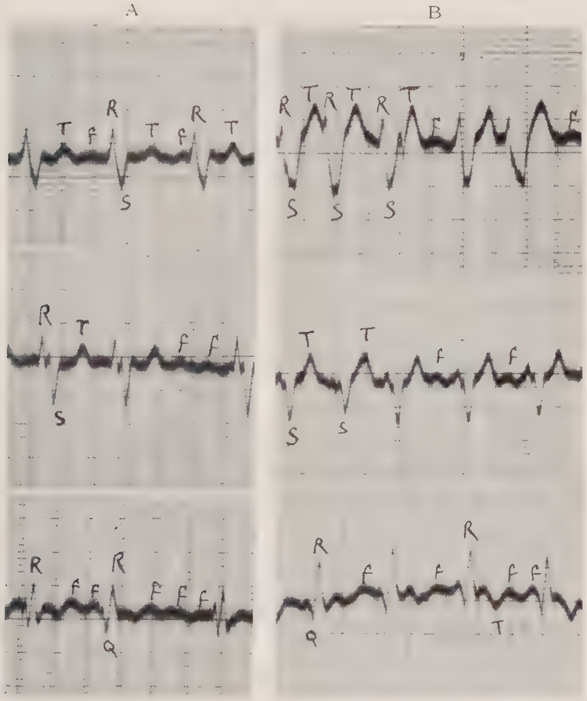

Fig. 19. Auricular fibrillation with ventricular complexes of a partial lesion of the left bundle branch. B is the later record and was taken during a period of decompensation with rapid heart rate. In A, though right axis deviation is shown by the direction of the waves of Q R S, yet the notching and increased duration of Q R S (.12 sec.) indicate a disturbance of the spreading of the contraction. In B the still greater duration of the Q R S group makes it plain that this is present.

Notching of the Q R S Group. Abnormal notching of an upward or downward limb of one of the waves of Q R S is a relatively common finding.[1] If the notching is slight and occurs during a quickly moving excursion it may be straightened out, as it were, into a sort of thickening or slurring of the side of an R or S wave. Notching is illustrated in Figures 38 A and C, 20 A and 16 A and B, while slurring can be seen in Lead 2 of Figure 32, in Figure 20 B and C, and in many other records.

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[1] It has been pointed out in Chapters I and II that normal notching of Q R S occurs in one, or rarely in two leads, but in neither of the leads will it be found upon a part of the curve near the top of a tall peak.

Often it is plain that the notching of Q R S in one lead occurs at the same time in the group as it does in another lead, or perhaps notching in one lead will be found at the same time as slurring in another.

These notches or slurrings occur at the same time instant in the course of the Q R S group in the different leads because they are due to the same disturbance in the production of the electrical potential within the heart. The abnormal production of potential, in its turn, is due to an interference with the normal spreading of the contraction wave by lesions affecting a large area of the Purkinje ramifications, or by the presence of a large focus of diseased muscle in the ventricular wall.

Lesions affecting only small areas of the Purkinje ramifications probably do not lead to the occurrence of notching or slurring, for the contraction is spread around and past them by the intact branches at a considerable speed. It has been shown by experimental work that such lesions as are caused by a knife-cut across the endocardial surface of the ventricle of the dog do not lead to appreciable changes in the ventricular waves, and particularly not to notching of the Q R S group, unless the main bundle branches are involved.

Usually, notching is accompanied by an increase in the duration of the Q R S group. If this increase is not present the Purkinje tissue is, in the main, performing its function properly and the notching is probably due to lesions in the ventricular muscle. When the duration of Q R S is increased, as in Figures 19 A and 20 A, B and C, it indicates either that damage is situated high up in the secondary branches of the A-V bundle, or that the diseased area of ventricular muscle is very large. In all likelihood, both of these conditions are present in such cases.

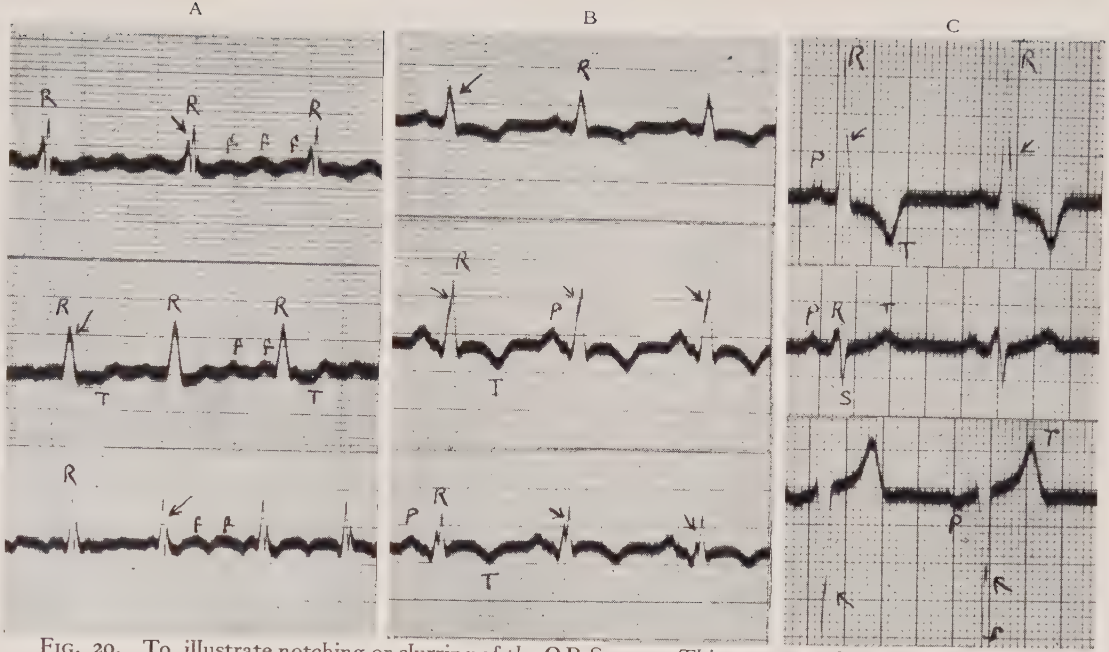

FIG. 20. To illustrate notching or slurring of the Q R S group. This occurs at the points indicated by the arrows. Record A also shows auricular fibrillation, Record B shows an abnormal T wave in Leads 1 and 2 and Record C shows left axis deviation of Q R S and an abnormal T wave in Lead 1.

A notched Q R S is very likely to have a downward T 1 or T 2 associated with it, especially if the duration of Q R S is increased. The abnormal inversion of T results from one of two causes: either the change in the time relations of the right and left ventricular contractions resulting from a large lesion of the Purkinje system, or the muscular imbalance of the two ventricles during the contraction when the notching is due to a large area of ventricular disease.

Duration of the Q R S Group. Increased duration of the Q R S group has been said to be due to a delay in the spreading of the contraction over the ventricular muscle. This may depend upon an increased thickness of the muscle of the left ventricle, or upon a dysfunction of the system of fibers which distributes the contraction stimulus to the ventricular muscle, i.e., the right and left branches of the A-V bundle, and the Purkinje fibers within either ventricle. If the stimulus is spread uniformly but slowly over the inner surface of the ventricles, there will be a prolongation of the Q R S group—an increase in its width, without any change in its form. A lengthening of the duration of Q R S occurs from this cause in some patients whose circulation has seriously failed, and in some whose heart rate has greatly increased. In the one case the cause is probably the defective circulation in the bundle-branch tissues, and in the other a too easy fatigability of the tissues. Figure 19 A and B are records obtained at different times from the same patient, showing an increased duration of the Q R S group with inreased heart rate, in a heart already affected by disease.

Localized disease affecting a considerable area of the Purkinje network or a large subdivision of the right or left bundle branch may lead to a prolongation of Q R S, because the area which should receive its stimulus by way of the diseased tissue is late in contracting. The amount of prolongation under such circumstances probably depends upon the size and situation of the area of ventricular muscle activated by the diseased conducting tissue. Such a pathology would very likely lead to notching of Q R S as well as a prolonged duration.

A shortening of the duration of the Q R S group is the normal change with increased rate, but it is found under a few other circumstances. In records from children Q R S may have a duration of only .06 sec., possibly because of the small size of the ventricles, so that the spreading is quickly completed. I have seen a very brief Q R S in a record from a heart which had just suffered from a coronary artery thrombosis which must have put a considerable area of muscle tissue out of function (Fig. 28 A).

Voltage of the Q R S Group. The height of the largest wave of the Q R S group seems to vary from much the same causes as does the height of P. It is usually large when there is an increased amount of muscle, whether or not the hypertrophy leads to a *preponderance* of one ventricle. It is larger when the physiologic state of the muscle is better, and smaller when this is not so good. The upper limit of normal has been set at 16 mm. and the lower at 7 mm.; but with a wave as large as R it is always necessary to add 10 or 15 per cent to the largest recorded value when one lead has a very small relative excursion. The reason has been explained in Chapter II. Under these circumstances then, the minimum recorded value might be less than 7 mm. and must be less than 16 mm. in order to lie within normal limits.

After the acute infectious diseases and during marked circulatory failure this excursion is found small. It increases during convalescence and with improvement of the circulation. Figure 21 A and B are records from the same patient, the first taken just as he was recovering from a period of severe cardiac failure, and the second three weeks later, after he had improved greatly under treatment. Note the increased size of the excursions in the second record.

Many patients with sclerosis and narrowing of the coronary vessels give records with the Q R S excursions of very small size, and these do not increase to normal size even when compensation improves. Figures 22 A and B and 28 B are from such patients. Record 22 B shows an interesting feature in the small excursions of the waves of the ventricular extrasystoles also. This is quite in accord with the theory as to the reduced size of the deflections of the normal beats. The muscle of this heart must be in a very poor physiologic condition.

Most patients with congenital cardiac abnormalities have very large excursions of Q R S (Fig. 23 c); also many with aortic regurgitation (Fig. 18 A) or marked mitral stenosis (Fig. 11 A) or high blood pressure (Fig. 11 B). In all these conditions the cardiac hypertrophy is the factor which causes the increased size of the deflections. The size of Q R S is not, however, governed by a single factor. A heart whose R wave would be very large if the muscle were in good condition may show one within normal limits or even less if the functional condition of the muscle is below par for one cause or another.

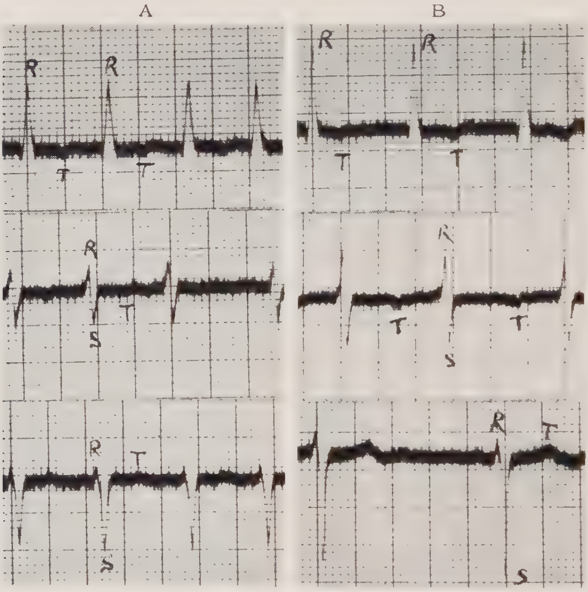

FIG. 21. Two records of the same patient, the first taken just after recovery from severe heart failure, and the second several weeks later, after he had further regained compensation. Both records show auricular fibrillation, both show marked left axis deviation of Q R S and both show a downward T wave in Leads 1 and 2. The difference lies in the size of the excursion of the waves of the Q R S group and of the T wave, Record B having much the larger excursions.

A varying state of nutrition of the heart muscle will lead to variations of the maximum excursion of Q R S, no matter whether the waves are normal, notched or of the type associated with bundle-branch lesions. It is not possible, however, to attach this significance to variations in the height of the waves from one time to another, unless the form of the wave remains the same to show that the contraction is spreading by the same path each time. Variations of the path of the contraction can cause variations in the height of the waves, as has been discussed under bundle-branch lesions. The appearance of notching in the Q R S group might be expected to be associated with a change in the height of Q R S.

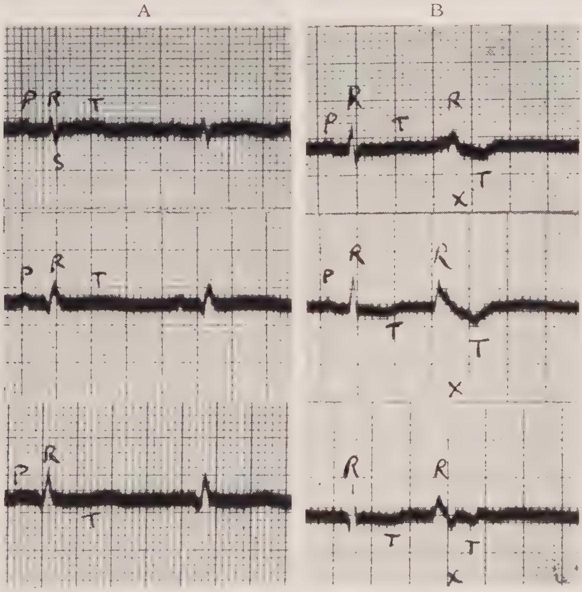

Fig. 22. Showing the very low voltage sometimes developed by the Q R S excursions. Notice that P and T are correspondingly small, as are the waves of the premature ventricular beat (X) in Record B. The patient of Record A showed these small excursions at every examination for a period of years.

Nor is it possible to attach much importance to slight differences in the height of the waves of records from different persons—to affirm that the muscle of one heart is in better or in worse condition than that of another. There are too many independent factors which can increase or decrease the voltage, e.g., the degree of hypertrophy, the path of the contraction, or the physiological condition of the muscle fibers. If the waves, however, are very small, under 6 or 7 mm., or very large, over 16 or 18 mm., we can feel safe in saying that the condition of the muscle of the first heart is poor, while that of the second heart is good. Compare, for example, the large excursions of the Q R S group of Figure 12 A with the small ones of Figure 22 A or B. These hearts have a different pathology, but the statement that the one giving the former was in good physiological condition was supported by the patient's relatively good compensation at the time the record was taken; and the statement that those giving the latter were in poor physiological condition was supported by the appearance of marked cardiac insufficiency. On the other hand, Figures 13 A and 17 A are two records of the same heart, the first taken when the patient was not well compensated, the second at a later period when the condition had become serious.

Voltage of T. There is a general correspondence between the variations in the height of Q R S and of T in the lead giving each its largest excursion. Most conditions affecting the ventricular muscle vary the size of these waves in the same direction, but some affect them differently. It appears that three factors can vary the height of T in its largest lead: (1) The structural integrity of the myocardium; (2) its state of nutrition; and (3) the strength of the ventricular contraction. A small T will result from a diffuse myocardial disease, but also from a poor state of nutrition of the muscle fibers and from a weak contraction. A large T will result from disease affecting the bundle branches or Purkinje system so as to cause notching of the Q R S group, and also from a strong ventricular activity, which of course demands a good condition of the muscle fibers.

To state a hypothetical comparison of the influence of these different factors, a heart without pathological processes in the ventricular muscle but with a poorly nourished muscle might give a small Q R S group and a small T. With slight diffuse pathology in the ventricles, such as might be due to arteriosclerotic narrowing of the coronary arteries (Fig. 22 A) or to the toxemia of an acute disease, Q R S might be normal in every way, and the T wave less than the normal size in its largest lead. The greater the involvement of the muscle, the smaller would be the size of the T wave. If a heart had a diseased ventricular muscle and was not in a good physiologic state, then both Q R S and T would be of small size; but if the disease should affect the bundle branch or Purkinje tissue, T might then be within normal limits in spite of the poor condition of the muscle. An abnormally small T should make us suspect the integrity of the ventricular muscle, even without any other electrocardiographic abnormality.

An abnormally large T, if not due to an abnormal path of the ventricular contraction, is a sign of a strongly contracting ventricle. It is found after exercise, during acute thyrotoxic states and with ventricular hypertrophy. Even the presence of these conditions will not produce a large T wave unless the muscle is both free from diffuse disease and in good functional condition.

Abnormal Direction of T. The direction of T should not be downward in any but Lead 3, and even here it is found downward in only 30 per cent of records from normal hearts. The direction of T in the three leads is changed by factors which affect the character of the ventricular contraction.

The T wave, like the Q R S group, has a normal and an abnormal direction for its electrical axis. The normal T with upward deflections in Leads 1 and 2 and either an upward or a downward one in Lead 3 has an axis which is shown in Figure 10 to lie between 0° and 90°. The range for its normal direction is greater than for the Q R S group. When T gives a downward deflection in Lead 1 or Lead 2 its electrical axis is of course abnormally directed but there is no especial significance as with the Q R S group whether it is deflected to the right or the left of its normal position.

In the case of premature ventricular beats and of bundle-branch lesions it is changed so as to be opposite to the chief excursion of the Q R S group in two leads, if not in three. Furthermore, these T waves are of unusually large size. The abnormal inversion of T in these records is due to the fact that since one ventricle starts its contraction before the other, it is also the first to start its relaxation. This earlier activity of one ventricle disturbs the electrical potential in such a way toward the end of the contraction that an abnormally inverted T wave results. It does not follow, however, that abnormal inversion of T is always caused by the precedence of the activity of one ventricle: certainly not when the Q R S is normal, for then neither ventricle precedes the other to an abnormal degree.

When Q R S shows abnormal notching or slurring, it is common to find that T is downward in Lead I or Lead 2 or both (Fig. 20 B and C). This change may be attributed to the effect of the basic pathology upon the character of the ventricular contraction. If the notching is due to a Purkinje lesion, the change in the direction of the T wave probably arises because a certain region of the ventricular muscle is late in contracting and therefore late in relaxing, just as occurs with a lesion of one bundle branch. If notching is due to a large myocardial focus, the changed direction of T may arise because the diseased area does not enter into contraction to the normal degree at its proper time. In the one case the abnormal T is due to an abnormal time relation of the events of the ventricular systole, while in the other it is due to an abnormal functional balance during the contraction.

Quite recently Wilson and Finch have shown that the T wave of the human electrocardiogram can be modified in its direction by drinking a large amount of iced water. This is ascribed to the cooling of the diaphragmatic surface of the heart which lies against the stomach. The T wave of experimental animals is similarly affected when localized areas of the ventricles are cooled by the direct application of such cooling agents as the ethyl chloride spray or the carbon dioxide pencil. Cooling depresses the physiological activities of the affected area, and we have here a demonstration that such depression can cause an abnormal inversion of T in an otherwise healthy heart.

When left axis deviation of the Q R S group is present it is common to find T directed downward in Lead 1 or in Leads 1 and 2 (Fig. 18 A). When right axis deviation is present T is often downward in Leads 2 and 3 (Fig. 23 A). This change in the T wave is similar to that which takes place when one ventricle precedes the other in its contraction because of a lesion of one bundle branch. The T wave lies opposite to the predominant direction of the Q R S group in each lead. In Chapter III this feature of the T wave of axis deviation curves was mentioned in detail, and it will suffice here to repeat the conclusion that this change of T is due to something other than the cause of the axis deviation. This other factor I believe to be a diseased condition of the

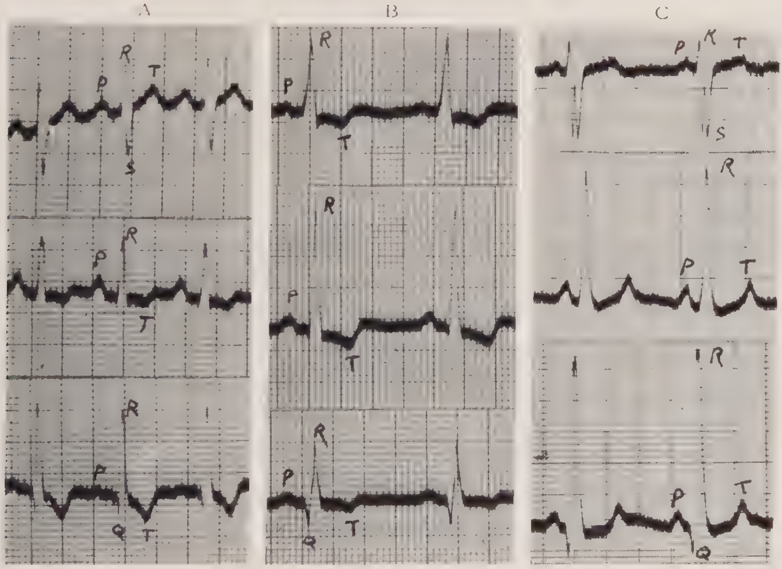

FIG. 23 A. The downward T wave in Lead 2 which frequently appears in records with right axis deviation of Q R S.

B. Showing normal direction of Q R S with the T wave turned downward in all three leads. This patient was not under the influence of digitalis. The Q R S group is abnormally wide, measuring .12 sec. in Lead 3.

C. A heart with congenital abnormality, showing the unusually large excursions of Q R S along with a notching and increased duration of Q R S that is rare in such records.

ventricular muscle, the direction of the deviation apparently determining whether T shall be inverted in Leads 1 and 2 or in Leads 2 and 3.

Myocardial changes may lead to inversion of T in all three leads with either right or left axis deviation, or even with a normal direction of the axis of the Q R S group. Inversion of T in one or two or in all three leads may occur in a record which does not show either right or left deviation of Q R S and in which Q R S does not have notching or slurring of its waves (Fig. 23 B). This change would be due to a diffuse myocardial process—either a toxic or an acute inflammatory or a chronic sclerotic process—which has not involved the bundle branch or Purkinje tissue so as to cause notching.

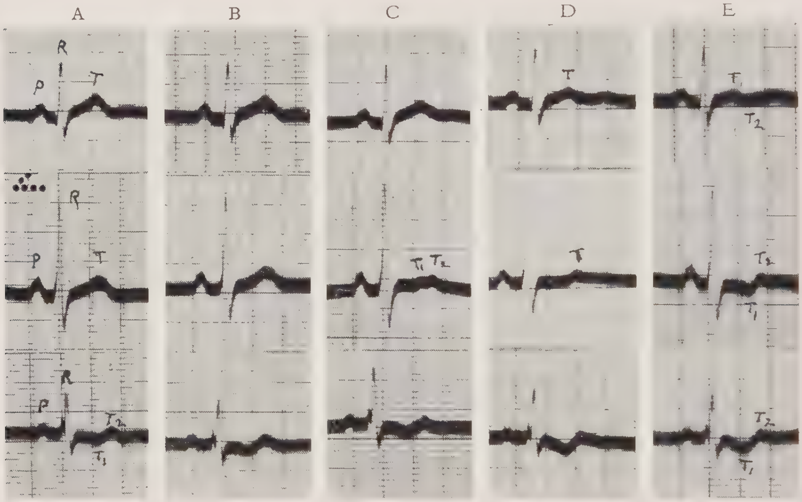

Fig. 24. Progressive increase of the digitalis effect upon the T wave. Note the peculiar double-topped T wave of Lead 2, Record C, and the difference in the behavior of the peak of T and of the portion of the curve immediately preceding it.

Ventricular disease is not the only cause of an abnormally directed T wave. The effect of the drinking of iced water has been mentioned. Toxic conditions as different as uremia and the acute febrile stage of trichinosis can also invert T. In both of these conditions I have seen a downward T wave which became normally directed after recovery from the acute condition. The poisons of pneumonia and typhoid fever do not have this effect upon T. Certain drugs affect T in this way. Especially noteworthy because of their common use in cardiac disease are digitalis, quinidine and morphin.

The *effect of digitalis* is very characteristic and usually consists of a diminution of the height of T with a depression of the S-T interval so that it comes to lie below the zero level. T becomes smaller and ultimately disappears into this increasingly large depression. The change of T can be traced in the serial records of Figure 24. Another type of change due to digitalis consists in a diminution of the height of T until it becomes zero in the lead which showed the smallest excursion, and then passes below the line, without any appreciable change in the level of the S-T interval. When right or left axis deviation is present the effect of digitalis changes the T wave in such a way that it finally comes to be opposite in each lead to the direction of the predominant wave of Q R S. The change does not always progress to this degree, but when it does the curve will have assumed the form described above for the combination of axis deviation of Q R S and myocardial disease (Figs. 23 A and 18 A).

The effect of digitalis on the T wave can be noted to a slight degree by the second or third hour after the administration of a single dose of the tincture equal to one minim per pound of the patient's weight. A dose of 50 minims will usually produce a slight change by the third or fourth hour. This effect upon T reaches its maximum for that dose about six hours after the drug is given, and does not diminish appreciably until after twenty-four hours. It passes off very slowly, and traces of a marked effect may persist for as long as three weeks after the drug is discontinued. For this reason final conclusions should not be drawn as to whether an abnormal T wave is due to disease until after the drug has been withheld for this interval. Figure 25 A and B are records of a patient taken ten and twenty days respectively, after a course of digitalis. It is seen that even in 25 B, the T wave shows some slight tendency to depression of the S-T interval in Leads 1 and 2. It is curious and noteworthy that digitalis has no constant effect on the Q R S group of the electrocardiogram. It is not the spreading of the contraction, but the contraction itself that is affected by the drug.

The effect of quinidine and morphin upon the electrocardiogram has not received such careful study as has the effect of digitalis. Both of these drugs tend to change the form of the P wave, probably by displacing the site of impulse formation from its normal place in the sinus node (Chap. VI). Morphin produces abnormal inversion of the T wave, as does quinidine also. The latter, however, causes a prolongation of the Q R S group and a great prolongation of T, which are evidences of the powerful effect of this drug in delaying the spreading of the contraction and the process of the contraction itself.

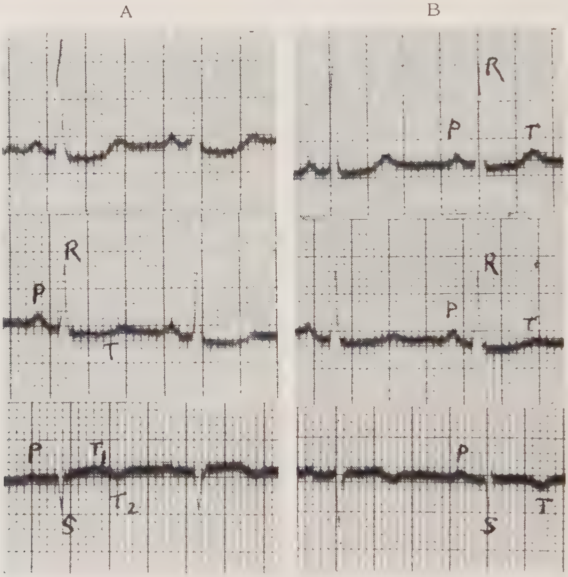

Fig. 25. Records of the same patient taken ten days (A) and twenty (B) after full digitalization. Note that the space between R and the peak of T is distinctly below the zero level of the record in A, and slightly so in B, showing that in the latter the digitalis effect has not yet entirely gone.

Notching of T occurs only rarely. This wave is typically a smooth rounded or peaked elevation, unless it should have a P wave superimposed upon it, as in Figure 30 and Figure 33 A and B. The only records I have ever seen suggesting a notched T wave are like those of Figures 11 b and 24 c. In these the notching appeared only after the administration of considerable doses of digitalis. Records of these patients before the drug was taken do not show this notching of T, and I can say nothing as to its significance.

Coronary Occlusion T Wave. Some patients show a special peculiarity of the T wave of the electrocardiogram. Five of these records are seen in Figures 26 and 27, the characteristic feature common to them all being the upward convexity of the S-T interval indicated by the arrows. The T wave in one or more leads is turned downward, often

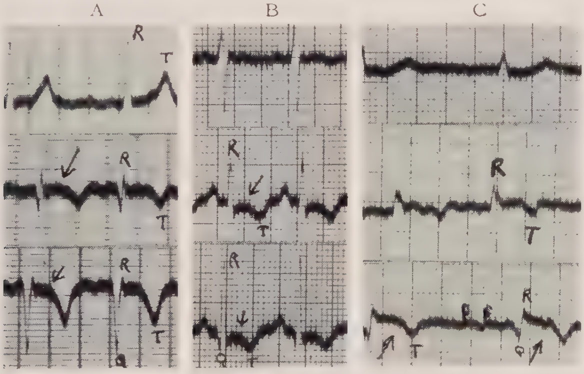

Fig. 26 a and b. Showing the peculiar upward convexity of the T wave that results from coronary artery occlusion. This curve is indicated by the arrows and is best marked in Lead 3. It is sometimes evident in Lead 2 also, as in a and b. Record c is an atypical form, but still recognizable.

sharply peaked and shows an upward convexity of the R-T or S-T interval. If this feature is found only in Lead 3 it cannot be considered significant unless T 2 is also turned downward. The upward convexity need not be present in this lead also. These records usually show some degree of left axis deviation, more rarely right axis deviation, and often have a large Q wave in Lead 3. Some of them have notching or slurring in the Q R S group (Fig. 20 b).

Figure 26 a is from a patient who had a typical attack of severe precordial pain, associated with slight dyspnea and collapse, and a very remarkable abnormality of the electrocardiogram (Fig. 28 a). He undoubtedly had an occlusion of a large branch of one of the coronary arteries. Figure 26 B is from a patient who, having had less dramatic though no less typical symptoms, was shown at autopsy to have had a recent occlusion of a coronary branch resulting in an area of degenerated musculature in the lateral wall of the left ventricle. Figure 27 B is from a patient of Dr. Herrick of Chicago. This heart proved to have a large area of degenerated musculature with replacement fibrosis in the wall of the left ventricle near the apex.

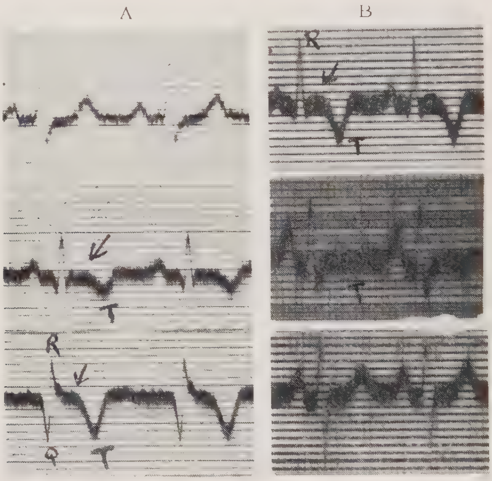

Fig. 27. Records showing same convexity of T as in Figure 26. Marked in Lead 3 of A, but present in Lead 1 of B. Evident also in Lead 2 of Record A. Record B was loaned by Dr. Fred Smith and Dr. J. B. Herrick.

The results of experiments on dogs support the idea that these abnormal curves have this origin, for after tying off a branch of the left coronary artery a practically identical curve is often obtained. The animal experiments would indicate that after occlusion of a branch of the right coronary artery, we should expect the record to take on a form suggesting right bundle-branch block. Possibly some of the human records which have this form have resulted from disease of the right coronary artery, but this has not yet been proven clinically.

Not only does this typical change in the T wave occur after sudden obstruction of the blood flow in a branch of a coronary artery, but a chronic narrowing which is especially marked in one branch can probably also cause it. Diffuse myocardial disease does not cause it because of the diffuseness of the pathology: a focal change is probably necessary.

This abnormality of T appears at some time after a coronary thrombosis or embolism, or is the result of a chronic narrowing. The curve immediately after the occlusion is quite different, as can be seen from Figure 28 A and B. The former is from the same patient who later gave the record of Figure 26 A and the latter from the one who later gave the record of Figure 26 C. Curves of this sort are obtained in experimental work also if the records are taken during the first few hours after tying off a coronary branch. The typical feature of these records is that the S-T interval does not start from the base line of the record, but comes off from the Q R S group at some point above the base line. I have not seen a record in which the S-T interval began below the base line, though such a record may occur, as it is conceivable

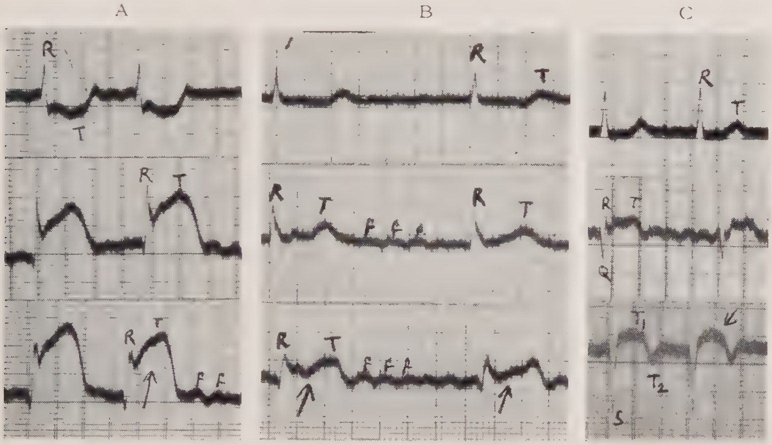

FIG. 28 A. Record from the same patient as Figure 26 A obtained very soon after the occlusion occurred.

B. Obtained very soon after the attack from the patient who later gave Figure 26 C.

C. Intermediate between Figure 26 A and 28 A, to show the changing position of the S-T interval and the T wave which takes place as the infarct heals.

In A and B the arrow indicates the peculiar elevation of the S-T interval above the base line, which is the early characteristic feature. In C the elevation of A is changing into the convexity of the S-T interval seen in the records of Figure 26.

on theoretical grounds. At a little later stage the S-T interval may lie above the base line and the peak of T may be downward, as in Figure 28 C. This record is from the same patient who gave Figures 28 A and 26 A, affording the transition between them, as can be readily appreciated.

The S-T Interval. Digitalis, as has been mentioned, has a very definite effect upon this portion of the electrocardiogram. Of course these changes are but temporary and pass off as the drug is eliminated. I have occasionally seen a record of a heart not under the influence of digitalis, which showed in more than one lead the peculiar change in the S-T interval produced by this drug (Figs. 24 and 25). This part of the record comes to lie on the opposite side of the zero level from the T wave so that the T wave is diphasic. It is possible that this is due to myocardial changes which if more marked would cause inversion of T. Occasionally a normal heart may show this feature to a slight extent in one lead in which T has a small relative value as Lead 3 of Figure 5 E, but never in more than one lead and never more than 0.5 mm.

In some records this level is found to be quite far from zero, perhaps 2 or 3 mm. above or below, and in the same direction as T. Deflections of over 0.5 mm. immediately after the Q R S group are rare and are probably abnormal, but we are quite unable to give a cause for this abnormality. It usually appears largest in records that show a marked axis deviation of Q R S combined with a large T wave. It may be that the contraction process develops this potential sooner than the peak of T, because of the greater activity of the hypertrophied ventricle. (See also Fig. 28 and the description of the record obtained immediately after coronary artery occlusion.)

Duration of the Ventricular Complex

The duration of the ventricular complex has been shown to vary with the heart rate. Besides this it has been suggested by experimental findings that if the diastolic filling of the ventricles is abnormally increased there will be an increased duration of systole. A ventricular complex, longer than the normal figure for the heart rate at the time of observation, might thus be attributed to veinous stasis and cardiac dilatation. For heart rates of 70 or over the maximum normal duration of systole may be set at .40 sec., measured from the beginning of the Q R S group to the end of the T wave. This suggestion has not yet been applied to clinical material.

There has also been a suggestion that an undernourished heart will have a prolonged systole, but though there are some clinical observations that might be interpreted in its favor, this hypothesis rests upon very insecure foundation. Digitalis has been observed to shorten the duration of the ventricular complex and quinidine to prolong it.

SUMMARY

We do not at present recognize any abnormalities of the P wave as being due to disease of the auricular muscle.

When the ventricular muscle is affected by disease, the Q R S and T waves are likely to undergo certain changes, one or more of which may be present in the same record:

1. Notching or slurring of Q R S.
2. Prolonged duration of Q R S.
3. A voltage of Q R S less than 0.4 or 0.5 millivolts.
4. T waves turned downward in Lead 1 or Lead 2 or both.
5. A voltage of the T wave of less than 1 millivolt.
6. A special peculiarity of T appears directly after a coronary thrombosis, the R-T or S-T interval coming off from Q R S at some distance from the zero level.
7. A different peculiarity of T is found at a later date after coronary thrombosis and in patients who have not had such an attack but who have symptoms to suggest chronic coronary narrowing. The T wave in one or more leads is turned downward and sharply peaked and the R-T or S-T interval is convex upwards.

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The T wave is affected by digitalis, quinidine and morphin and possibly by other drugs.

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CHAPTER V

CLINICAL SIGNIFICANCE OF ABNORMAL WAVES

A normal muscle is subject to functional variations from time to time and a diseased muscle is more liable to such variations. This is especially so if the disease progresses or diminishes. The height of the waves will vary from time to time with the physiological condition of the heart muscle. The duration of the P-R interval and of T will vary with the heart rate and with the functional state of the tissues concerned in their production. Such abnormalities as notching or abnormal direction of the waves depend upon an abnormal process of muscle contraction which is usually due to disease of the muscle, rarely to poisoning by drugs or toxins. The changes in the electrocardiogram due to structural disease are more likely to be permanent than the changes due to other causes, but it is quite possible for the changes from an acute disease to disappear entirely.

In order to decide upon the gravity of abnormal electrocardiograms we must know whether the abnormality is purely functional or is due to disease. The line of thought to be followed in deciding upon this is, on the whole, much the same as that for determining the significance of a murmur at one of the valve areas of the heart. Just as the presence of certain murmurs indicates definitely the presence of valvular disease, so does the presence of certain electrocardiographic abnormalities indicate definitely a diseased state of the muscle. When other abnormalities are found in the record their significance is doubtful and must be decided from the general features of the case, such as a history of a possible cause of cardiac disease, the presence of any sign of the heart failing in its function of carrying on the circulation (cardiac insufficiency) or the presence of such a physical sign of cardiac abnormality as enlargement, murmur or irregularity. If any of these things are found, it is likely that the abnormal electrocardiogram is due to disease. If all are absent we must conclude that if any pathological process is present it must be so slight that the compensatory powers of the heart have been able to overcome the handicap imposed thereby.

Neither the electrocardiogram nor any other single feature of the clinical examination is able, alone, to give the prognosis. If two hearts had the same abnormalities in the record there would still be a difference in the prognosis, depending upon the condition of the valves, the height of the blood-pressure, the character of the life led by the individual, etc. Yet the electrocardiogram is a very important feature, for it gives us the only direct evidence of disease of the ventricular muscle. Two patients who were exactly alike in all respects except their electrocardiographic records would still be far from similar if one had normal ventricular waves, while the other had an abnormal record like any of those of Figures 12, 14, 16 or 20. The one with the normal waves would have a much better prognosis, for his ventricular muscle is more likely to be free from disease.

Notching of the Q R S group is usually caused by heart muscle involvement from which there is no recovery. It is apt to be a rather diffuse myocardial disease, and if the notching of Q R S is persistently associated with small-sized waves or with an abnormally inverted T wave we may feel certain that the myocardium is extensively damaged. Patients showing this combination of abnormalities in their records have almost without exception a greatly limited cardiac reserve, and do not improve so as to become even approximately normal. Willius has shown that the average expectation of life is distinctly less for patients with notching of Q R S than for patients otherwise similar, but with normal ventricular waves.

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Notching of Q R S depends upon a pathological process either in the muscle of the ventricles or in the ramifications of the Purkinje system. When in the latter situation, the disease may vary in extent or severity from time to time and there may be variations in the notchings or in the direction of the waves as a result. A variation in the notching may also be due to marked variation in the height of the waves of Q R S. Large excursions tend to stretch out the notches, as it were, and to make them less noticeable. Small excursions have the reverse effect, as may be seen in the records of Figures 13 A and 17 A which are from the same patient. The notching signifies the same change in the ventricular muscle, whether it is less noticeable in higher waves or more noticeable in smaller ones.

Notching may vary from beat to beat in some records in a cycle that coincides with the respiratory movement. This will be readily understood to be due to a rotation of the heart by the movements of the diaphragm. It is quite independent of cardiac function.

Those patients who have an electrocardiogram showing *bundle-branch block* tend, as a class, to have a considerably impaired circulation. The abnormal waves result from a disease which is usually extensive and of the chronic sclerotic type. Exceptions to this general statement do occur, however, for even a very small lesion in the ventricular muscle, if it happened to be properly placed, might affect the function of a bundle branch so as to cause this abnormal curve. Rarely patients are seen who have typical bundle-branch block complexes and yet are able to perform an average amount of exercise without signs or symptoms to suggest that their cardiac reserve has been seriously taxed. Figure 12 A is from such a patient. He has no evidence of valvular disease or cardiac enlargement. He has been under observation for four years and only lately has begun to show signs of failing cardiac power. These complexes of bundle-branch block sometimes appear during pneumonia or some other infectious disease. The record of Figure 14 B appeared during pneumonia, and during the following year changed to the form of Figure 16 A. During this year the patient, who also was free of valvular disease and cardiac enlargement, had a moderate limitation of his cardiac reserve, whereas, previous to his illness, he had been a very powerful man. The change is usually a permanent one, the bundle branch failing to recover its function and the abnormal complex persisting.

Variations in bundle-branch block complexes are not uncommon, and may be due to temporary variations in function in parts of the diseased bundle branch. Occasionally, in patients who developed the condition during an acute illness, complete recovery has taken place, or the complex has changed to one showing a less radical disturbance of the mechanism of ventricular contraction, e.g., notched Q R S with an abnormal T or normal Q R S with abnormal T. This event would indicate a temporary disturbance of the bundle's function, perhaps due to slight infiltration with round cells or some other process which could be removed. The prognostic importance of such a change in the waves would depend upon the danger of later damage by scar tissue formation. In the majority of cases, however, patients with curves showing bundle branch block are much limited in their ability to exercise, and do not improve to a condition of more than average comfort.

The T wave described as due to *coronary artery occlusion* has been found almost without exception in patients whose exercise was limited by precordial pain, even though the pain might have been so slight as to be described as merely a discomfort. Some of them have severe attacks of typical angina pectoris. Some have died of such attacks. Some few, when not hindered by the pain, have an abnormal amount of dyspnea on exertion. With these it is probable that in addition to the focal necrosis resulting from coronary occlusion, there is an extensive myocardial disease. The patients are often able to be about and carry on their work in spite of the pain and the dyspnea. I know of one who is still living, and with but slight limitation by his pain, more than four years after this electrocardiographic peculiarity was discovered. A patient with such a lesion has but little chance to recover more than a moderate amount of cardiac reserve, and there is the ever-present possibility that a large branch or a main artery may become stopped with a fatal result. I have observed instances of a return to normal from this form of the T wave, the patient improving greatly coincidently. I take it as evidence of a compensatory increase in the collateral anastomoses which exist between the small branches of the coronary arteries so that the anemic muscle area is again properly nourished. Others have also noted this occurrence.

Abnormal inversion of the T wave may result from the action of digitalis or morphin, so that the action of these drugs must be excluded before T wave abnormality can be ascribed to myocardial abnormality. If necessary we must obtain another record after these drugs have been excreted from the body. There are instances, as with uremia, which make it appear that the T wave may be inverted in Lead 1 or Lead 2 by the action of toxins arising in the body. These records sometimes show small excursions of the waves as well. The inversion disappears and the waves become larger after the cause of the abnormal function is removed, be it drug or tissue poison. If an abnormal inversion of T is accompanied by notching of the Q R S group the change is usually a permanent one. It is usually due to a myocarditis which is likely to result in changes of a fibrotic character, and therefore to be permanent.

When no cause for a muscle intoxication is present, we must consider an abnormal T to indicate a diffuse myocardial disease. The extent of this disease may be inferred by observing whether the excursion of the T wave is of good size and whether the Q R S group shows notching or slurring in the course of its waves. The character of the heart's response to effort will also help in this decision, if taken in conjunction with the presence or absence of valvular disease or a blood pressure of 200 mm. or more, for we must realize that the effect of either of these conditions may be too serious a handicap for even a fairly good heart muscle.

A heart with an abnormal T wave is a poorer risk than one without it, though if this is the only abnormality revealed by a thorough examination of the heart the patient is not likely to be more than slightly incapacitated. Over a period of years such a heart would tend to gradual failure. The follow-up reports of the Mayo Clinic show that when associated with other abnormalities of the cardiovascular system, T wave abnormality added considerably to the gravity of the prognosis, such patients having a shorter average duration of life than those with normal T waves. The finding of a downward T 2 and T 3 was less serious in this respect than if T 1 or T 1 and T 2 were downward.

The voltage of Q R S may vary from time to time with the functional state of the heart muscle, but without disease the variation keeps within the limits described in Chapter II. Moreover, such variations in height are only significant when the form of the Q R S group remains the same from one observation to another. A variation in the height of R or S is only significant if there is no change in notching of the waves. A large excursion of Q R S may result from the process which causes notching, as evidenced by the large size of the Q R S excursion of ventricular extrasystoles, and of the complexes of bundle-branch block. In the absence of notching, abnormally large excursions are a sign of a muscle that is hypertrophied. Small excursions of Q R S, e.g., between 12 and 6 mm., may be due to a subnormal functional state of the muscle. If smaller still, they are usually due to a diffuse fibrosis of the muscle. Patients with these very small Q R S groups are seldom able to carry on a normal physical activity, and are likely to continue to be thus restricted.

The voltage of T has a somewhat different significance from the size of the Q R S group, as is evident from the fact that both Q R S and T are not always large or small in the same record. A large T may be present because the muscle is normal and is contracting strongly, or may be due to hypertrophy of the ventricles. It may also be due to the change in the ventricular contraction produced by a lesion of the bundle-branch tissue with notching of Q R S. The size of T, therefore, must be considered in relation to the Q R S group.

In the same way, an abnormally small T is of itself an uncertain indication, but must be considered in relation to

Q R S. If this is also small in all leads the smallness of T may be due to a weak ventricular contraction; but if Q R S is normal or large an abnormally small T would indicate a diffuse fibrotic change in the muscle. This change is not usually severe enough to incapacitate the patient seriously, but it is rare to find a small T wave with large Q R S excursion from a heart with a normal reserve power. This myocardial condition rarely improves; though, as has been said of an abnormally inverted T wave, the course need not be rapidly progressive. If there is a notching of Q R S, the tendency to produce a large-sized T is such that even extensive myocardial disease may not produce an abnormally small T unless the heart is in extremis.

The finding of these electrocardiographic changes is of great value in helping to decide upon the presence of disease in the ventricular myocardium. With the qualifications detailed above we may be sure that when these abnormalities of the record are found, the muscle is diseased. The different abnormalities are not to be considered so much as the result of different degrees of myocardial involvement as of a different location or distribution of the process.

On the other hand, we are not able to say that whenever we find *a normal electrocardiogram* we are dealing with a normal muscle. A single small lesion which happened to involve one of the bundle branches would cause a marked change in the record, while if the same lesion were in the deep layers of the ventricular muscle it would probably not affect the record at all. Single small foci are an uncommon pathological finding in the heart muscle, and if they do occur are probably of little immediate clinical importance because of the large reserve force of the heart. We have had so few autopsy reports from patients with normal electrocardiograms that we cannot be positive as to the amount of ventricular disease which may be present without affecting the record in some of the characteristic ways. Those hearts that I have personally examined, 11 of which were reported with Masters, give the impression that a normal electrocardiogram is not obtained from a heart which has more than slight localized disease of the ventricular muscle; an amount which can be determined only by careful microscopic examination, and which would not be expected to affect appreciably the cardiac reserve power. Nor would such disease be expected to increase to any extent a handicap from some extra-muscular cause, such as valvular disease.

This opinion is in accord with clinical experience. It is an uncommon event to find a patient with symptoms of cardiac insufficiency who does not have either an abnormal electrocardiogram or some other demonstrable cause of cardio-respiratory embarrassment such as valvular or marked respiratory disease, or high blood-pressure. The difficulties of such clinical observations must be emphasized, for the cardio-respiratory functions are so interdependent that disease in different organs may cause a similar clinical result. No one case will prove any of the points above discussed. A series of cases with one feature, such as abnormal T wave or notched Q R S, in common, will show an average clinical picture which may be considered as, in part at least, the result of the special feature. It is from observations of this kind that the opinions herein expressed have been reached.

Chapter VI

DISTURBANCES IN RATE OR RHYTHM

Our knowledge of the disturbances in the rate and rhythm of the heartbeat has made great advances in the last ten years. First the polygraph and then the electrocardiograph was used to investigate the mechanism of their production, and a tremendous volume of facts has been accumulated. The electrocardiograph has proved far superior to the polygraph in this work, because it not only gives the time relations of the contractions of the auricles and the ventricles, but indicates by the form of the waves when the contraction passes normally over these chambers and when it does not.

Sinus Arrhythmia

The simplest disturbance of the cardiac mechanism is an irregularity which arises from unusual variations in the activity of the vagus. This is called sinus arrhythmia because the vagus acts upon the sinoauricular node (the sinus node) which originates the normal heartbeat, retarding or accelerating its activity according as the vagus is more or is less active. The contraction stimulus starts at its normal place in the sinus node, and passes normally throughout auricles, A-V node and bundle to the ventricles. The auricles and ventricles, therefore, contract normally and the form of their waves is normal. Their rhythm however, is irregular because the sinus node originates its impulses irregularly.

The most common type of sinus arrhythmia is due to an increase of vagus activity with slowing of the heartbeat during the expiratory phase of respiration and a decrease of vagus activity with acceleration of the heart during inspiration, as seen in Figure 29 a. A slight degree of this sort of irregularity is very common.

Almost any record will show variations of .02 to .04 sec. in the intersystolic interval, but even more marked variations are by no means uncommon, especially with the heart rate under 80; especially also in children, and in people over fifty years of age. In Figure 29 A the rate variations can be seen to coincide with the variations in the Q R S group due to the movements of the diaphragm. In another form of sinus arrhythmia the variations in vagus activity extend over longer periods than the respiratory cycle (Fig. 29 B) perhaps lasting even ten or twenty seconds to each phase. The ventricular irregularity is only secondary, for each beat follows the auricular beat in the usual way after a normal interval. It is the auricular beats themselves that are primarily irregular. A characteristic feature of the electrocardiogram with sinus arrhythmia is that the waves do not change along with the arrhythmia. The ventricular waves may be of normal form or may show any sort of abnormality. Whatever they may be they *do not vary throughout the record.* This is also true of the P waves, for the sinus node continues to originate auricular contractions in the normal way, and the spread of the contraction wave is unaffected by the vagus activity. In this feature, sinus arrhythmia differs from premature contractions of either auricular or ventricular origin, for these are associated with an abnormal site of origin of the contraction in the chamber which is premature, and with an abnormal form of the electrical curve of this chamber.

Certain rare records of sinus arrhythmia will be seen to show a slight variation in the form of P coincident with the variations in rate. This is more usual in the type with long phases of slowing and acceleration than in that which follows the cycle of respiration. It is seen to a slight degree in Figure 29 A and is due to the vagus activity displacing the site of impulse formation from the more irritable upper portions of the sinus node to the less irritable lower portions. As the node is from 3 to 5 cm. in length, this would cause the impulse to enter the auricle in a different place from normal and would thus change slightly the path of the contraction wave.

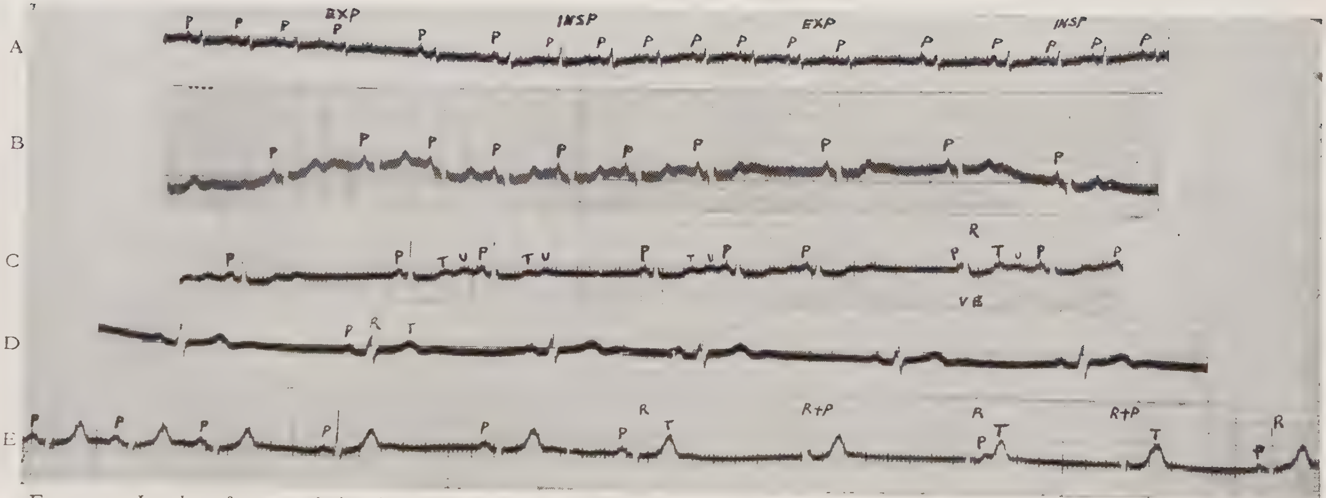

FIG. 29 A. Lead 3 of a record showing sinus arrhythmia. The P waves always precede the Q R S group by the same interval, but the interval between successive P waves continually varies.

The phase of respiration is marked at the top of the record, and it can be seen that the rate variation follows this closely.

Note also the variation in the height of the R waves with the respiratory phase, these being larger at full inspiration and smaller at expiration.

B. Lead 2 of another record of sinus arrhythmia, the phases of slowing and acceleration being quite independent of respiration.

C. Sinoauricular block. The long pauses between auricular waves will be found to be exactly twice the interval between the P waves at the faster rate.

The seventh ventricular complex is an instance of ventricular escape. The preceding pause is so long that the ventricular irritability cannot tolerate it, and so this beat comes before its time, beginning just after the peak of the delayed P wave.

D. An instance of slow heart (37 per minute). There is also a sinus arrhythmia.

E. An instance of sinus slowing followed by ventricular escape. The 7th, 8th and 9th ventricular contractions begin before the respective P waves, but the 10th follows P after a normal interval.

Another curious and rather uncommon example of vagus activity is seen in what is called *sinoauricular block.* It is evidenced by the appearance of a normal series of auricular and ventricular waves from which occasionally one complete heart cycle will be missed, the next one following after an interval almost exactly double the normal interval (Record C of Fig. 29). Such doubling of the interval occasionally persists for a time, leading to a halved heart rate. The mechanism of this dropping of a heart cycle is supposed to be a physiological blocking of the impulse which normally passes from the sinus node to the auricle, so that this fails to receive a stimulus from the node and accordingly does not beat until the next stimulus arrives.

Some individuals have a persistently slow heart rate, less than 70 per minute, especially men of the athletic type. Certain ones have rates considerably less than this when at rest, perhaps 55, or 50, or rarely 40 per minute, and their heart rate does not increase with exercise to the same extent as in those with a more normal rate when resting. The electrocardiogram with this slow rhythm is quite normal in every way except in the matter of the heart rate. Figure 29 D is a record from such a patient, the rate being 37 per minute—as slow as found with heart-block. As the P wave and the ventricular waves are quite normal in all three leads, showing a normal path of the contraction wave and therefore a normal heart mechanism, it is proved that the slowed heart rate is due merely to a slowed stimulus production in the sinus node. This slow heart action, like sinus irregularity, arises from overactivity of the vagus, and we often see in such records a slight variation in the diastoles, so that sinus arrhythmia is also present. This is the case in the figure. In certain older patients, especially in those past fifty years, the slow rate may be so very persistent that it seems as though some local disease condition had lowered the irritability of the node. Perhaps a deficient blood supply to the sinus node because of arterial narrowing might do this.

Figure 29 E shows another occasional result of excessive vagus activity. The auricular activity is so very slow during the latter part of the record that the ventricles beat without an impulse from the auricles, being unable to tolerate such a long rest. This phenomenon of *ventricular escape* is a very interesting demonstration of the heart's inherent tendency to beat. The ventricular complexes have, throughout, the form which is normal for the individual of this record, indicating that their contraction stimulus is received along the normal paths. This is usual when the auricular stimulus fails, for the rhythm-producing function is taken over by the auriculoventricular junctional tissues, probably by the A-V node. The contraction impulse is thus normally distributed over the ventricles, and produces the normal ventricular complex of the individual. Figure 29 C also shows ventricular escape in the seventh ventricular complex.

Rarely there will be periods of considerable duration when the auricles are inhibited in this way, so that the junctional tissues may take over the rhythm-producing function, but at a slower rate than normal. When this "nodal rhythm" is in effect, it is usual for the auricle to contract in response to a stimulus which is considered to have passed back from the A-V node to the auricles. The P wave falls either with or just after the Q R S group and has an abnormal form, usually inverted, because of the abnormal path by which the stimulus enters the auricles. The eighth P wave of Figure 29 E evidently does not arise in this way, for it has the normal upward form, and therefore was originated in the sinus node. In this record there is not "nodal rhythm."

The term "ventricular escape" has been applied to certain instances which showed a normal ventricular complex occurring at a slightly more rapid rate than that of the auricles. This seems to me to be an obvious misnomer, for the term "escape" in this sense could be as well applied to obviously abnormal rhythms in which the ventricles are faster than the auricles, such as ventricular tachycardias. These cases with ventricles more rapid than auricles should be considered as tachycardias of nodal origin, for they are evidences of an overirritable ventricle rather than of an underirritable auricle, as is the case in the classical "ventricular escape."

Premature Beats

The next most frequent disturbance of the cardiac mechanism is due to premature beats, sometimes called extrasystoles. These give rise to an iregularity of the ventricular systole and may produce only a very small pulse or none at all, depending upon how much blood has passed into the left ventricle since the preceding systole; that is, upon the degree of the prematurity. A premature beat may originate in any part of the auricles or the ventricles, or in the auriculoventricular system which connects these parts of the heart. It is conceivable that the sinus node could originate premature beats, but such an occurrence has not come to my attention. In any case the electrocardiogram will always show very plainly the part of the heart from which the premature beats arise.

Auricular Premature Beats. The diagnostic feature of this condition is the premature occurrence of a P wave of different form from the others of the record in question. It is due to a contraction originating at an abnormal focus within the auricles at a time previous to that when the normal stimulus enters the auricles from the sinus node. The abnormal focus is called *ectopic* because it lies outside of the sinoauricular node where the normal impulses originate. This *ectopic* contraction, starting at an abnormal point, spreads over the auricles by an abnormal path and produces an abnormal P wave. The abnormal form of P enables us to recognize a beat as ectopic even if its prematurity is only a matter of a few hundredths of a second.

Often, as in Figure 30, the first manifestation of abnormality of the curve may be a distortion of the T wave of an otherwise normal ventricular group, by the premature P wave falling with T. This occurs in all three leads of this record. If the auricular beat is not so early it will be plainly seen in the diastolic interval, as in Lead 2 of this record. The second premature beat in this lead can be observed to have a P wave of different form from the normal P waves of this record, and yet to follow sooner after the preceding T wave than do the normal P waves. If the P waves of the other premature auricular contractions of this record could be separated from the T wave and considered individually, they too would be found to be different in form from the normal P waves of this case. The normal P of this record is upward, broad and notched in Leads 1 and 2, while the premature P which is superimposed on the T is not so high or broad in Lead 1, and is downward in Lead 2. Note also the abnormal forms of the various premature P waves of Figure 33 A and B.

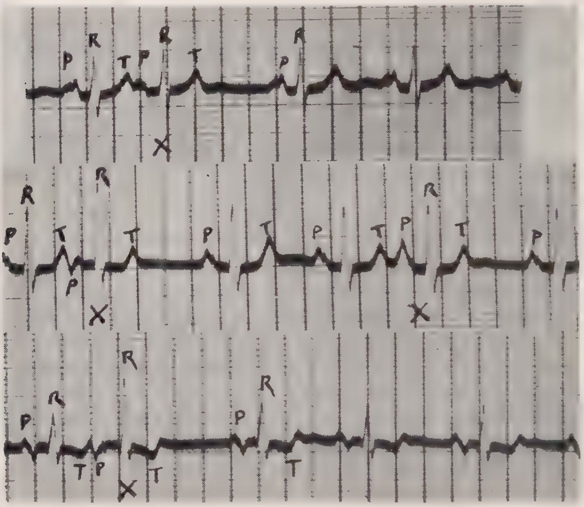

Fig. 30. Three leads of a record showing auricular premature beats. The premature P waves are at the points marked X and have a different form from those of the other auricular contractions. There are two premature contractions in Lead 2, each from a different auricular focus.

In each instance in the illustrations the ventricular complex follows the premature auricular beat after a normal P-R interval. The conduction time after these premature auricular beats is a severe test of the ability of the A-V conduction system, for the rest period since it last functioned is much shorter than usual. A latent defect in the conduction system will sometimes be made evident only by the fact that a premature auricular beat is followed by a prolonged conduction time, or by a complete failure of ventricular systole, due to a complete blocking of the impulse.

There is one circumstance under which a premature auricular beat may fail to be followed by a ventricular beat without reflecting on the integrity of the auriculoventricular system. When the auricular beat is so premature that the impulse from it arrives in the ventricles before they have relaxed from the preceding systole, then they fail to respond to it. The ventricles are refractory to stimuli while contracting, so that this impulse from the auricles fails to cause a ventricular contraction even though it has passed normally along the bundle. This is why the P waves during ventricular escape are not followed by another ventricular beat. The auricular stimulus arrives when the ventricles are still contracting (Fig. 29 E).

The ventricular complex following the premature auricular beat always has approximately the same form as the other ventricular complexes of this individual, but *always* it is *slightly* different from the others, as can be seen in Figure 30. This difference is evident in records showing the auricular premature beats in three leads. The curve may be unchanged in any one lead, and yet quite a variant in the other two. Both Q R S and T are changed, though as a rule Q R S is more evidently so. The change in the Q R S group depends upon an abnormal spreading of the contraction in the ventricles, and this causes a change in the contraction itself, which to a greater or lesser extent affects the T wave.

Lewis has suggested that the change in Q R S is due to a slight delay in the conduction along either the right or the left branch of the A-V bundle, or to a delay in conduction along some of the finer arborizations of the bundle branches upon the inner wall of either ventricle. He has called these contractions, arising in this way, *aberrant contractions*, and the ventricular complexes to which they give rise, *aberrant complexes*. This aberration, he believes, occurs after premature auricular beats, because their prematurity does not allow the conducting tissues the usual rest period before they are again called upon to function. Such an explanation would point to a slight abnormality of the A-V system at some localized area, or at least to an abnormal physiology in one branch of the bundle. It is true that, according to our theories of the electrocardiogram, such a delay in conduction would cause the type of changes in the ventricular complexes which we find in these *aberrant contractions*. From the clinical viewpoint, however, it is difficult to believe that *all* patients who have premature auricular contractions have an abnormality either of function or of structural disease in the A-V conduction system; and it is curious that these aberrant complexes are so rare when heart-block is present with dropped beats, a condition which we know to be associated with disease in the A-V system. Moreover, aberrant complexes are common in normal dogs with artificially induced premature auricular contractions, and it is difficult to find a reason why there should be a localized abnormality of the A-V system in normal dogs even when under an anesthetic.

A better explanation of this aberrant feature may be found in the abnormal character of the premature auricular beat itself. The contraction does not involve the auricles in the normal way, and so may affect the A-V node abnormally. As a result of this the impulse may fail to pass down the two branches of the bundle simultaneously, as it should, and the resulting asynchronous stimulation of the two ventricles would result in the change in the ventricular complex.

The pause after a premature auricular beat, before the next auricular systole supervenes, is always at least slightly longer than the normal intersystolic interval for the record, but is seldom long enough entirely to *compensate* for the prematurity of the beat which started the disturbance. The space of the two intervals, from the last normal auricular contraction to the premature contraction and from this to the next normal auricular contraction, is nearly always less than that of two normal intervals in the same record.

If a record is found which shows a premature P wave of normal form in all three leads, and if the interval between this and the next P wave is *equal* to the normal interval, then this premature auricular beat must be considered to arise in the sinus node. This post-extrasystolic pause, however, has always in my experience been found to be greater than the normal interval between beats. The increase in the duration has been explained as due to the remoteness from the sinus node of the focus starting the premature beat. The longer the post-extrasystolic pause, the more remote the focus.

Ventricular Premature Beats. When premature beats arise in the ventricles they can be recognized by the appearance of waves which have certain distinctive features, shown in Figures 31 and 32. There is first a large upward or downward wave representing the spreading of the contraction—the Q R S group—which usually shows notching or slurring on one or both of its sides or at its peak. This wave lasts for .14 to .20 sec. and is followed by a wave that looks like the ordinary T wave, but is much larger. It is always directed opposite to the large Q R S excursion, except in a lead in which the initial deflections are unfavorably recorded,[1] as in Lead 2 of Figure 31 B. In this small lead the T wave may be diphasic, or absent, or in the same direction as the largest wave of Q R S in this lead, or in the opposite direction. In such small leads the Q R S group is very often notched or has both upward and downward waves.

These complexes are not usually preceded by any indication of a P wave; or if they are, as in Record A of Figure 32, it is at so short an interval that it is plain that A-V conduction had not taken place before the onset of the ventricular contraction.

An auricular wave occurs at the proper rhythmic interval after the preceding P wave, and may fall with the Q R S group of the premature beat or with the T wave of this complex. It can be seen as a slight notching or deformity of the curve of the premature beat in Lead 3 of Figure 32 B. This submerged P, though sometimes quite plain, is sometimes scarcely visible, especially if it falls during the Q R S group (Fig. 31 A). The next P wave following the premature beat comes at the proper interval from the submerged P, and is followed by a normal ventricular complex; the rhythm of the auricles is unaffected. The pause after the premature beat, the post-extrasystolic pause, is *compensatory*, in the sense that it is sufficiently longer than the usual diastolic pause of the record to *compensate exactly* for the prematurity of the beat. The time between this post-extrasystolic and the preceding *normal* ventricular contraction exactly equals two normal intervals. In Records C of Figure 31 and C of Figure 32 there are no P waves, because auricular fibrillation is present, and there cannot be a compensatory pause.

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[1] Showing a small height in proportion to the height of the waves of Q R S in the other leads.

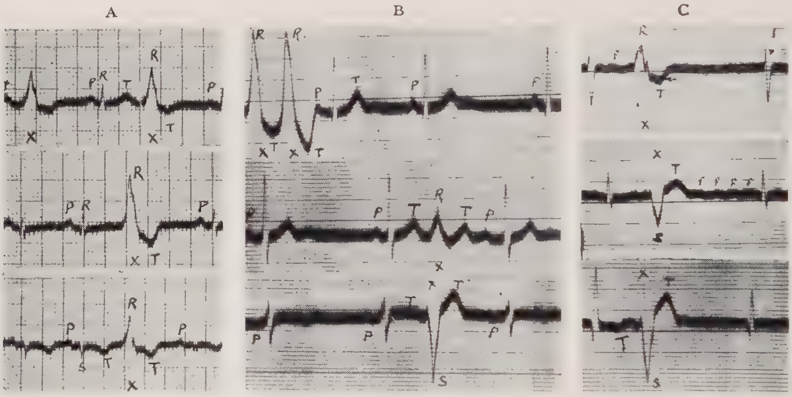

FIG. 31. Three records of ventricular premature beats by the three leads. The premature beats are marked X.

Records B and C have premature beats of the type considered to arise in the left ventricle.

The other ventricular complexes of Record B indicate a slight left axis deviation of Q R S; those of Record C a definite right axis deviation. Record C also indicates that auricular fibrillation is present.

In a patient with a slow heart rate a premature ventricular beat may occur and be completed before the next contraction stimulus arrives from the auricles; thus the premature beat is truly an *extrasystole*, being *interpolated* between two normal beats. This occurs in Record B of Figure 31, which record also shows a sinus arrhythmia. Such *interpolated* beats practically never result in a pulse wave, as they are too premature. The ventricular filling is so incomplete early in diastole that the blood ejected by the systole does not cause a pulse of appreciable size.

The ventricular complex of premature beats arising in the ventricles has an abnormal form for the same reason that the P waves of premature beats from the auricles have an abnormal form. The contraction wave arises in the ventricles at an abnormal point. It is ectopic, and therefore spreads over the ventricles in an abnormal manner. It is because of this abnormal form that we are able to recognize these premature beats in the presence of auricular fibrillation (Figs. 31 C and 32 C) a rather difficult distinction in polygraph records.

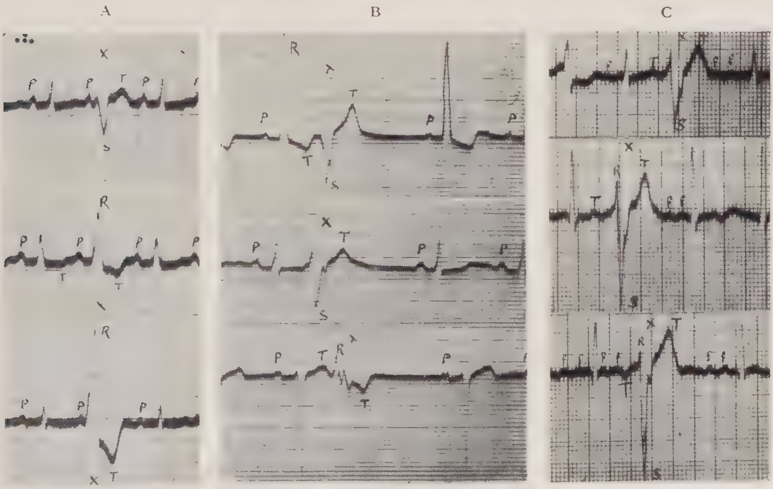

Fig. 32. Three records of ventricular premature beats by the three leads. Records A and B show the type considered to arise in the right ventricle. The other ventricular complexes of Record B indicate a marked left axis deviation of Q R S; Record C indicates auricular fibrillation.

The increased duration of the Q R S of these beats arises as does the prolonged Q R S of bundle-branch block. It is due to a delay in the spreading of the contraction over the whole heart, which in turn is due to its spreading from one ventricle to the other through the muscle of the septum or around the lateral walls of the heart. The stimulus of the premature beat starting in the muscle probably passes first to the Purkinje tissue of the ventricle, involves that ventricle in the contraction, and then, having spread through the septal muscle to the Purkinje tissue of the other ventricle, extends through this to the ventricular muscle on this side.

The relative size and direction, in the three leads, of the Q R S group of these premature beats, varies greatly; and it is possible to determine, from the direction of the waves in the three leads, the ventricle in which the beat originates. The sign indicating the ventricle first affected is a deviation of the axis of Q R S similar to that produced by preponderant hypertrophy of one ventricle. Hypertrophy of one ventricle impresses the electrical effect of that ventricle upon the electrocardiogram by dint of the increased electrical capabilities of its increased muscle fibers. A premature beat starting in one ventricle gives that ventricle a predominance in time which results in impressing its electrical effect upon the curve before the other ventricle becomes fully active.

The type of curve produced by a beat starting in the right ventricle is seen in Figure 32 A and B. It shows right axis deviation of the Q R S group, with the chief deflection of Q R S downward in Lead 1 and upward in Lead 3, while Lead 2, though usually upward also, may be either downward, or small, or may have large R and S deflections. The T wave of these curves is upward in Lead 1 and downward in Lead 3, which is opposite to the Q R S deflection in those leads, while in Lead 2 it takes a direction opposite to the largest peak of Q R S, or may be practically zero, or diphasic, first downward then upward. The left ventricular type of premature beat is shown in Records B and C of Figure 31. It shows left axis deviation of Q R S with the initial deflection upward in Lead 1 and downward in Lead 3 with T opposite in each case, while Lead 2 will be the same as described for the curve of beats originating on the right side.

The evidence for the localization of these beats is from the results of experimental work on dogs and monkeys, in which premature ventricular beats were produced by mechanical or electrical stimulation of the ventricles. Such stimulation has caused curves of the type described with great uniformity in monkeys, but in dogs some exceptions are found, probably owing to the special structure of the ventricles of the dog and to their position in the thorax.

Electrocardiographic records have been obtained of the premature beats produced by mechanical stimulation of the human ventricle. Since the heart has always been covered by the chest wall of the patient it is difficult to be certain of the exact points stimulated but the evidence seems to be in agreement with the localization to right and left ventricles as stated above.

The numerous combinations of directions which the Q R S may take in different patients indicate that such a division into beats of right and left ventricular origin cannot be more than approximate. In 45 patients of the author's series in whom the premature beat was obtained in 3 leads, the initial deflections were found as follows:

	Per Cent	
− − −	7	
− − +	5	R 33 per cent
− + +	28	
+ + +	20	
+ + −	12	L 40 per cent
+ − −	28	

Note. Deflection + = upward; − = downward.

Although the division into right and left ventricular types is indicated by the brackets, there are other beats, illustrated in Figure 31 A and Figure 32 C, which cannot be so classified. These probably originate in the interventricular septum or very close to it.

Ectopic ventricular beats are believed to be usually initiated in the Purkinje fibers, though probably never in fibers which are far from the junction with the ventricular muscle. Less often, they may arise in the ventricular muscle itself. The curves of such beats as are produced by stimulation of the ventricular muscle in animals are much notched in the initial phases, and these much-notched curves, though they do occur in human records (Fig. 31 c) are relatively rarer than the curves with smoother upward and downward movements.

Though it is undesirable to enter into a controversy in such a book as this aims to be, it is perhaps allowable to mention that this localization of the origin of premature beats in one or the other ventricle is being challenged. Einthoven is the first exponent of the idea that such a beat as shown by Record c of Figure 31 would originate at a point "more toward the right than the left half of the heart, more toward the apex than the base, and more toward the lower than the upper half of the heart." Such a point would lie toward the right near the apex of the ventricles ("rechts bei der Herzspitze"). Considering the curves by this method, one can mark out a sector upon the heart within whose limits the contraction arose. These sectors usually contain parts of both right and left ventricles. This method of localizing the source of ectopic ventricular beats does not disagree with the experimental results from dogs as reported by Rothberger and Winterberg, who, however, did not mention it. Also in agreement with this hypothesis is the fact that human hearts have been reported by Oppenheimer and the author as showing lesions in the right and left bundle branches, from the first of which was obtained an electrocardiogram of the type of Record c, Figure 31 and from the second, one of the type of Record A, Figure 32 (Chap. IV, bundle-branch lesion).

The evidence at present is conflicting, as can be seen, and indecisive. The burden of proof must rest with Einthoven and his followers, who attempt to overthrow a general belief. It is the author's opinion, in spite of the pathological report just mentioned, that it will not be overthrown.

Clinically the problem is of course unimportant, for even with its bearing on the localization of bundle-branch lesions (q. v.) it is of little concern to the physician whether the ventricular contraction starts in one or the other ventricle, or even whether the ventricular disease is more on the right or the left side of the septum. Our clinical interest centers on the fact that ventricular systoles are being initiated independent of the impulses from the auricles, and that the region which initiates these systoles is an over irritable area in the ventricles themselves, or at least in the Purkinje strands below the branching of the auriculoventricular bundle.

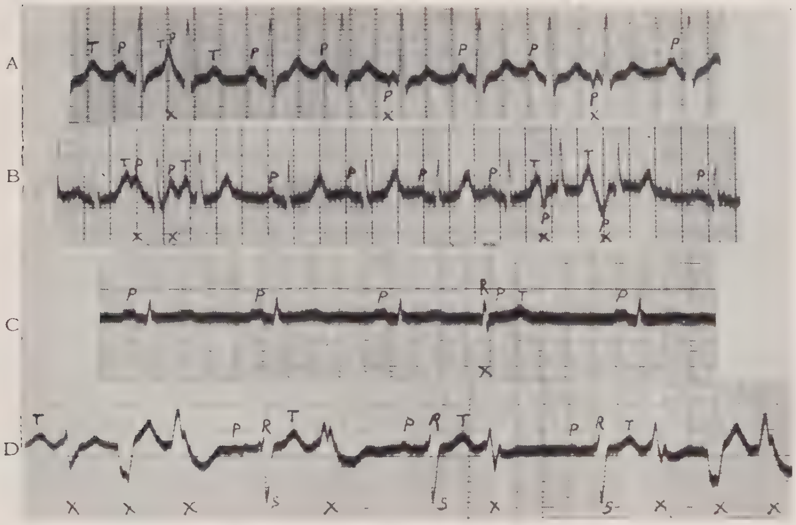

FIG. 33 A. Auricular premature beats from three different foci. Note the different forms of the different premature P waves.

B. Two pairs of premature auricular beats, each pair from a different focus. Note that each P wave of a pair is like the other of this pair.

C. A premature beat arising in the A-V node. The P wave occurs at the proper rhythmic interval after the preceding P wave and falls between S and T of the premature beat. It appears to have the same form as the other P waves.

D. Premature ventricular beats arising in several foci in succession. The first three complexes have the features typical of beats starting in the ventricles, and are each different in form. The fourth beat is the normal for this patient. It follows a P wave with a slightly prolonged A-V conduction time. The fifth beat is again of ventricular origin and from still a different focus. The sixth beat is again *supraventricular* and the seventh is still a different ventricular *ectopic* beat. The last three beats are of ventricular origin, but repeat the form of some of the previous beats.

Nodal Premature Beats. A premature beat may arise in the auriculoventricular node or in the auriculoventricular bundle above its branching. When this occurs (Fig. 33 C) no auricular wave will be found before the ventricular complex and this complex will show by its normally brief Q R S group that the ventricles have been stimulated together as normally. Lewis has introduced the term "supraventricular impulse" to apply to a point of origin for the stimulus which is above the branching of the auriculoventricular bundle. Such an impulse produces a ventricular complex of the supraventricular form like the normal for this individual, though slight variations may occur, as in the aberrant complexes of premature auricular beats. Aberrant ventricular complexes are probably here, as there, due to slight predominance in time (precedence) of one or the other ventricle, owing to the impulse passing more freely into one bundle branch than into the other. In the case of the nodal premature beat the following pause is compensatory as for a ventricular premature beat, because the auricles are not affected by the premature contraction.

Multiple Premature Beats. Sometimes premature beats from different foci will be found in one record. Figure 30 and Figure 33 A and B all show premature ectopic P waves of more than one form, and Figure 33 D shows several forms of premature ventricular complexes. This indicates two or more foci of increased irritability in the auricles or the ventricles, as the case may be, and points to a more extensive and therefore more severe myocardial affection.

Premature beats from any focus may occur rarely, or, as in some of the records of these figures, quite frequently. The more frequently they occur, the greater the irritability at the focus. Sometimes two or three ectopic beats from the same focus may occur successively, as in Figure 33 A and B or Figure 31 B. This denotes a great local irritability and is closely allied to paroxysmal tachycardia. If different foci originate sequential ectopic beats, as in Figure 33 A, B and D, the conditions are ripe for fibrillation of the auricles or the ventricles, as the case may be.

Sometimes, as in Figure 33 D, there will be a condition of overirritability of several foci, which may make the ventricles beat as rapidly and irregularly as they do when auricular fibrillation is present.

HEART-BLOCK

Heart-block is a depression of the function of auriculoventricular conduction. It may cause various sorts of disturbances of the heart action. If slight, it only lengthens the time that elapses between the auricular and ventricular contractions and cannot be recognized without instrumental examination. If more marked, it leads to irregular heart action, with dropped beats at the heart and in the pulse; but if complete, the heart is again regular. The rate of the ventricular contractions may lie anywhere between 22 and 90 or more per minute, a fact not always clearly realized. A slow heart rate does not justify a diagnosis of heart-block, nor does a rapid rate prove that it is not present.

Complete heart-block is always due to disease of the junctional tissues, as are probably the lesser grades of block also.[1] The disease may affect either the auriculoventricular node or the bundle before its branching. Some authors have attempted to place overactivity of the vagus in a causal relation to heart-block, but it is extremely doubtful if it ever produces more than an accentuation of a condition fundamentally due to disease. In my opinion, a clear instance of vagal complete heart-block has not yet been reported.

In Figure 34 A the P-R interval (P-Q R S group, more properly) is longest in Lead 2 where it occupies .28 sec. The normal P-R interval does not exceed .20 sec., and in children or adults with the heart rate over 90 per minute it should not be as long as this. If the patient is strongly under the influence of digitalis, we may find a P-R interval of .28 sec. without disease being present—perhaps more. It is rare to obtain a figure over .24 sec. with a normal bundle. Figure 34 B is an example of the effect of digitalis. This patient's P-R interval had been .20 sec. and was increased to .32 sec. after digitalis had been given. This degree of prolonged conduction would strongly suggest disease of the bundle, even considering the fact that digitalis was contributing to the result.

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[1] Intraventricular block is a term applied to the results of disease affecting the branches of the bundle, i.e., bundle-branch block, and bears no relation to the condition commonly called heart-block.

Digitalis will never affect a normal bundle so as to produce the grade of heart-block with dropped beats. With this there is occasional failure of the auricular stimulus to arrive in the ventricles, so that a ventricular beat is lacking. This is seen in Figure 34 c where the P-R interval progressively lengthens in Lead 1 from .22 to .32 or .40 sec. and the next P wave fails to be followed by a ventricular complex. The gradual lengthening of the conduction time is a result of increasing fatigue of the bundle, so that eventually it fails to function. The next succeeding P-R interval after the rest is again .22 sec. Such dropped ventricular beats usually occur irregularly, as in this record. They may be very infrequent, or may occur

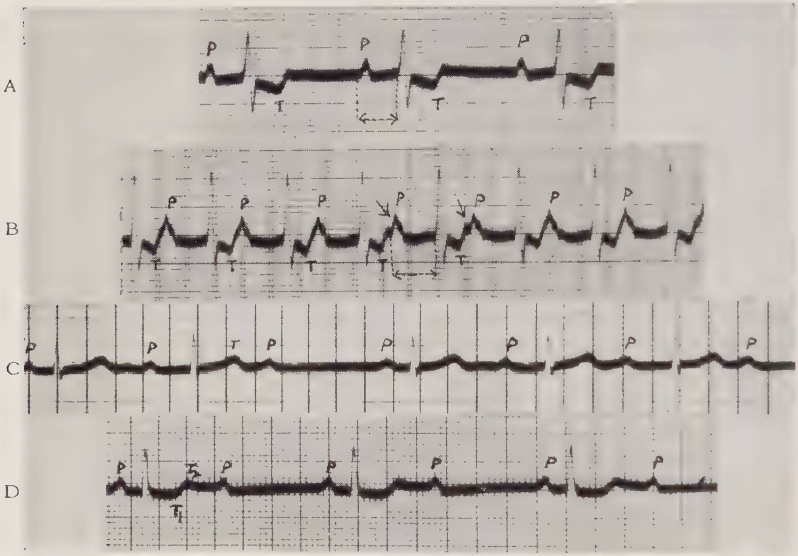

Fig. 34 a. Record by Lead 2 to illustrate a prolongation of the A-V conduction time (P-R = .28 sec.).

b. A marked prolongation of the A-V conduction time so that the P wave falls with the preceding T wave (P-R = .32 sec.). As the rate varies slightly the overlapping of P and T is more or less complete.

c. Showing the dropped beat phenomenon with increasing conduction time before the conduction fails.

d. The dropped beat occurs regularly after every other auricular beat, so that the ventricles beat at one-half the auricular rate.

after every second or third auricular beat, quite regularly for a time. It is typical of this degree of heart block for the frequency of the dropped beats to vary from time to time, so that both rate and rhythm of the ventricles are variable.

It is interesting to note the differences in the behavior of the A-V conduction system in different hearts that are diseased, and in the same heart at different times. Sometimes there will constantly be a long conduction time, as in Figure 34 B, without the occurrence of dropped beats; again a dropped beat will occur in the cycle following one with a comparatively short (.28 sec.) conduction interval. In one heart I obtained long records with a conduction interval of .60 sec. without a dropped beat, and then suddenly the dropped beat phenomenon appeared as in Figure 34 C with a conduction time varying from .28 to .40 sec. Shortly after this the heart again became regular; but records were not taken.

When dropped beats occur after every other auricular systole, the ventricular rate will be regular and will be just half the auricular rate, so that it will usually vary from 35 to 60, depending upon the rate of the auricles. Record D of Figure 34 shows this condition, the ventricular rate being 40 per minute and the auricular 80, with a conduction time of .20 sec. This rhythm will sometimes persist unchanged for many hours.

A feature of the electrical curves of heart-block which must be emphasized is that the form of the waves from beat to beat is not changed by the abnormal cardiac mechanism. The P wave is usually of normal form, or if abnormal it retains the same abnormality throughout. The ventricular waves are often normal. They may show right or left axis deviation or some other abnormality in form, but with a rare exception, to be detailed later, they do not vary from beat to beat. Note especially that even when the conduction time is varying the ventricular waves do not show "*aberration*" such as we find after premature auricular beats.

When the P wave coincides with the larger ventricular waves it becomes submerged and may be difficult to make out. Usually, a comparison with other ventricular groups will enable us to note a deformity which can be identified as due to the P wave because it occurs at the proper rhythmic interval. The P wave is especially difficult to make out when it falls with the T wave of the beat preceding, as in Figure 34 B. This super-position is rarely found in all three leads of a record, so that a comparison of the time of onset of the P waves in the different leads, measuring backward from the beginning of the Q R S group, will usually serve to decide which movement of the line of the record is P. Lead I of Figure 34 B served to analyze this record by the plain separation of P and T, though the notching at the point indicated by the arrow in the lead of the figure would suggest a separation between T and the following P wave. This notching occurs with variable plainness because of a slight sinus arrhythmia, which makes the interval between beats longer at some times than at others, and in the longer pauses separates P from the preceding T wave.

The next increase in the severity of heart-block leads to a rather uncommon condition. There is an incomplete dissociation between the auricles and the ventricles, so that the stimulus comes through to the ventricles only occasionally. For the other beats the ventricles contract with a slow regular rhythm which usually arises in the upper portions of the auriculoventricular system, above the branching of the bundle. We recognize this as the place of origin for these beats because the ventricular waves have the proper supraventricular form of the patient in question. If the stimuli originated below the branching of the bundle, and this occasionally has been observed, the waves would resemble ventricular premature beats. These supraventricular contractions resulting from stimuli within the auriculoventricular node or bundle, are always *aberrant*, that is, different from the complexes which result when the stimulus comes from the auricle in the normal way in the same heart. The difference probably depends, as in the case of the aberrant curves following premature auricular beats, upon a varying pathway through the auriculoventricular bundle.

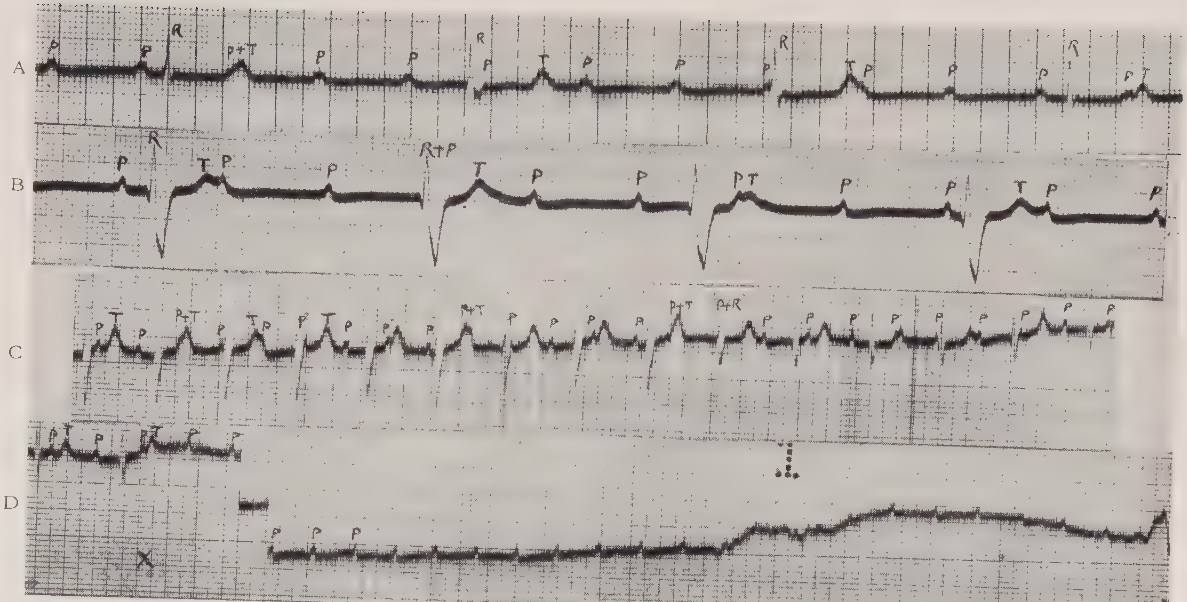

FIG. 35 A. Complete A-V dissociation. The auricles beat regularly 90 times a minute and the ventricles regularly 27 times a minute.

B. Complete A-V dissociation, the ventricular complexes showing an abnormal form.

C and D. A continuous record of a patient who was having Adams-Stokes syndrome with convulsive attacks. The ventricular beats stopped at X, the auricles continuing to beat to the end of the strip. The movement of the record at Y was due to the onset of a convulsion, fourteen seconds after the last ventricular systole.

A much more common event than this is a complete dissociation between auricles and ventricles, as shown in Figure 35 A. The P waves are seen to be regular at a rate of 89 per minute, the ventricular waves being 26 per minute and also regular. The ventricular waves in this figure have the characteristic supraventricular form; Figure 35 B shows complete heart-block with the ventricular complexes of abnormal form and therefore probably not of a supraventricular origin.

Occasionally, when the block is complete the form of such abnormal ventricular waves will vary from beat to beat. There is usually also a slight coincident irregularity in the ventricular rhythm and both variations are considered due to a varying site of the impulse formation. If the intersystolic interval should be regular we would consider the phenomenon due to variable conduction of an impulse arising in the A-V node through one or the other of the bundle branches. Premature beats occasionally arise in the ventricle when complete heart-block is present, interrupting the slow regular flow of stimuli.

Auricular fibrillation may be present coincident with complete heart-block, as in Figure 42 B. This will make no difference to the ventricles, which are removed from auricular influence by the block and which will continue to beat slowly and regularly unless there should be premature beats or a varying site of the origin of the stimulus. The effect of partial heart-block upon the heart with auricular fibrillation will be discussed under the latter heading. It causes a slow but irregular ventricular action.

The electrocardiographic records can cast a very interesting light upon the mechanism which precedes and accompanies an attack of Adams-Stokes syndrome. C and D of Figure 35 are parts of a continuous record, and show the heart action in one of these cases. The condition, at the beginning of Record C, was complete heart-block with an auricular rate of 96 and a ventricular rate of 54 per minute. The ventricular complexes first were of the right ventricular type, but later the contraction of the left ventricle was less delayed, and the complexes resembled the normal ones of this case. There were frequent variations from beat to beat, however. Suddenly at X the ventricles ceased to beat—their automatic center failed to function—the auricles meanwhile continuing to beat as before. After 15 sec. had elapsed the patient had a general tonic convulsion during which the ventricles again started to beat from a focus within themselves and the cycle was repeated. This patient had convulsion after convulsion about six times an hour. The attacks were always preceded by cessation of the heart sounds, and the ventricular beats always resumed. It is probable that the irritability of their automatic center was enhanced by the asphyxia to a point at which it again gave rise to contractions.

In other cases, when incomplete heart-block is present, the mechanism during the Adams-Stokes attack is different from this. A partial block suddenly becomes complete and a period of 5 to 8 sec. may elapse before the auricular stimulus again passes to the ventricles or the ventricles take on the function of originating the rhythm (ventricular escape). Such attacks are usually not accompanied by convulsions, but only by pallor and perhaps loss of consciousness.

Tachycardia

A tachycardia which might be called physiological, in that it is a simple acceleration of a normally contracting heart, is recognized from the electrocardiogram by the P wave and the ventricular complex having the normal form for the patient in question, or having only such slight changes as may be the result of the rapid rate. The changes noted might be short duration of P-R, Q R S and S-T and a slight reduction in the size of the waves. Figure 36 A is a record of such a tachycardia, the rate of auricles and ventricles being 150 per minute. Figure 37 A also shows simple tachycardia with a rate for the auricles of 150 per minute. Owing to the presence of lowered function of the A-V bundle there is present in this record a 3:2 heart-block and the

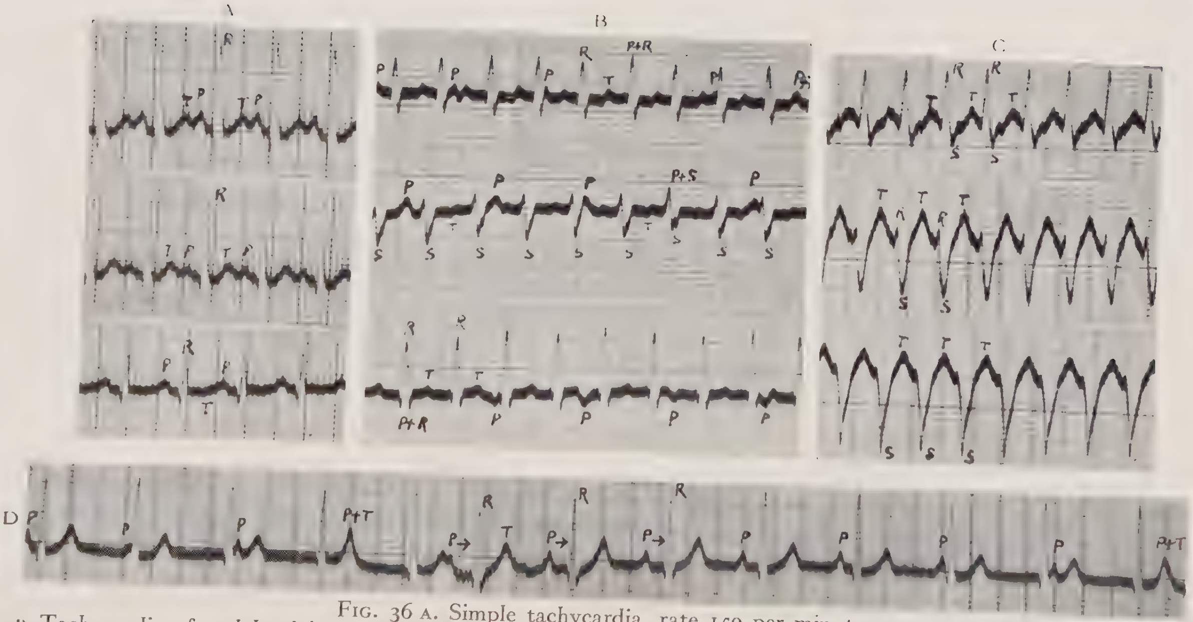

FIG. 36 A. Simple tachycardia, rate 150 per minute.
B. Tachycardia of nodal origin (A-V node) combined with complete A-V dissociation. Auricular rate 103 per minute, ventricular rate 180 per minute.
C. Tachycardia of ventricular origin (left ventricle). The rate is 215 per minute.
D. Tachycardia originating in the A-V node, but having a rate little faster than the auricles. Auricular rate 84 per minute; ventricular rate 90 per minute. For three heart cycles in the center of the record the auricular beat is followed by the ventricular in the normal manner, proving that the A-V conduction function is not disturbed. The next ventricular beat follows P at a shorter interval and probably originates from the A-V node, as do the later ones. Note the aberrant ventricular complexes of the nodal rhythm. They have no S wave and T is not so high.

ventricular rate is irregular, giving coupled beats and a rate of 97 per minute. When the auricular rate became more rapid in this patient the heart block became 2:1 so that the ventricles were regular at 80 per minute. When the 3:2 rhythm was in effect the typical lengthening of the P-R interval could be seen, as in the figure, in the two beats before the block. This combination of a simple tachycardia and partial heart-block is not common.

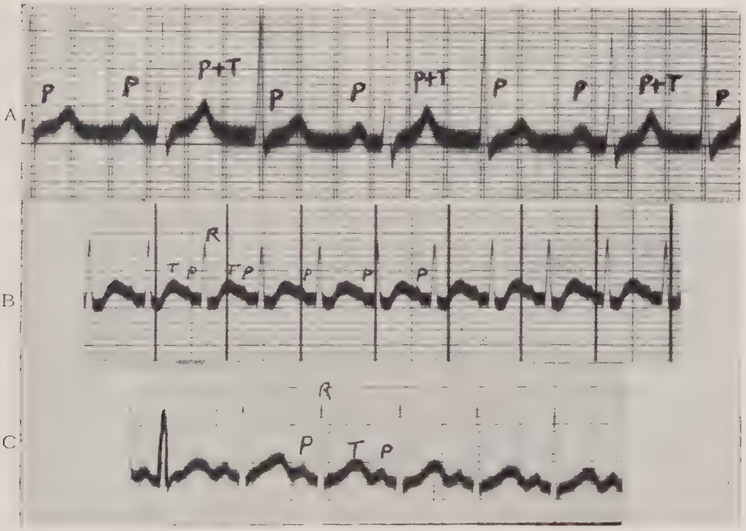

FIG. 37 A. Simple tachycardia combined with partial heart-block and the dropped beat phenomenon. Auricular rate 150 per minute. Ventricular beats occur in couples, rate 100 per minute.
B. Paroxysmal auricular tachycardia with auricular rate 195 per minute.
C. Later record of the same patient by the same lead. The rate is now 145 per minute, a simple tachycardia. Note the different form of the P wave in these two records.

The rate of these physiological tachycardias increases gradually at their beginning and declines gradually when the rate slows. In this they are distinctly contrasted to what might be called the pathological tachycardias which are paroxysmal in type, starting and stopping abruptly, the rate remaining practically constant during their course. The rate of the physiological tachycardia is seldom found to be above 150 per minute, while the paroxysmal tachycardias usually, though not always, have much higher rates. Any rate over 150 per minute should be regarded as a probable paroxysmal tachycardia unless a record has proved it to be physiological, or unless exophthalmic goiter is present. The latter may cause very rapid physiological heart rates.

Paroxysmal tachycardia may have a rate as slow as 100 per minute or less, or as fast as 210 per minute or more in different cases. It is the result of the activity of an ectopic area of increased irritability, just as with premature beats. It is, in effect, a rapidly recurring series of premature beats which takes control of the heart rhythm. Paroxysmal attacks of rapid heart action which are due to auricular flutter or auricular fibrillation should not be called paroxysmal tachycardia, but only those attacks due to the regular activity of ectopic foci as described.

The point of origin may be in any part of the auricles or ventricles or in the A-V junctional tissue, just as with premature beats. The attacks last for a period of only a few heart cycles or for several minutes or hours, depending upon how long the irritability of the focus is so increased that it continues to originate beats. Figure 33 B shows two successive premature auricular beats which from the similarity of their P waves can be considered to come from the same focus. Figure 31 B shows two successive premature ventricular beats from the same focus. From a physiological point of view two successive premature beats constitute a short attack of paroxysmal tachycardia, but attacks to be clinically recognizable must last for several seconds at least.

Auricular tachycardias nearly always show a P wave of an obviously abnormal form, and always one of an abnormal form for the individual in question. The focus which originates the impulse is ectopic, so that the auricles contract in an abnormal manner and produce a P wave which is abnormal for this person. In Figure 37 B the auricular wave appears as a notch upon the descent of the T wave because of the rapid heart rate of 195 per minute. This P wave does not appear very abnormal on first sight, but if contrasted with that of Figure 37 C, which was obtained after the paroxysm had subsided, it will be seen to be different. Notice also that the ventricular waves of Figure 37 B are distinctly "aberrant" as compared with those of the normal rhythm of Figure 37 C. The ventricular waves are "aberrant" during an attack of auricular tachycardia, just as they are after a single premature auricular beat.

Figure 36 B is a record of a *nodal tachycardia*, the impulse originating in the A-V node. In this record there is also a complete dissociation between the auricles and the ventricles, the former beating 103 times per minute and the latter 180 times. Sometimes a nodal tachycardia may have a rate not much faster than the auricles, and whenever the impulse from the auricles finds the ventricles in the diastolic resting state it forces a contraction which slightly disturbs the regularity of the ectopic rhythm. This condition has been erroneously described as *ventricular escape*, and is illustrated in Figure 36 D where the auricles are 72 and the ventricles 90 per minute. The ventricular complexes have a typical supraventricular form, showing that the impulse arises from the A-V junctional tissues. The P waves are seen to bear no constant relation to the ventricular waves. Heart-block is not present in this record, for at the points indicated by the arrows the auricular impulse has caused a ventricular contraction after a normal conduction time. All other impulses from the auricles found the ventricles still contracting and therefore refractory to stimulation. Note that the ventricular complexes of the nodal rhythm are "aberrant" as compared with those which are due to the normal impulse. They lack an S wave and the T is not so high.

The ventricular complexes of Figure 36 B are also "aberrant" as compared with those which were found between the attacks, the latter showing less left axis deviation, a briefer Q R S group and an inverted T 3. We recognize this rhythm as being *nodal*, by the fact that the ventricular complexes have a typically supraventricular form with a duration of the Q R S group within normal limits.

In certain rare records of nodal tachycardia there is an inverted P wave following the Q R S group at an interval of .18 or .20 sec. from its beginning. In these cases the auricles are stimulated by an impulse conducted backward from the rhythm-producing center in the A-V node. This constitutes a true "nodal rhythm" for the whole heart.

Figure 36 c shows a tachycardia which arises in the left ventricle itself, a *ventricular tachycardia.* The P waves are entirely lost in the large excursions of the ventricular waves which have the typical wide, notched Q R S and the large T wave directed opposite to the chief deflection of the Q R S group, indicating an origin in the ventricular tissues or in a branch of the A-V bundle. They resemble the complexes of the premature ventricular beats of Figure 31 c which arise in the left ventricle. Ventricular tachycardias are much less frequent than those of auricular or nodal origin, but paroxysms have been reported showing right-sided and others left-sided forms of the ventricular complexes.

At the end of a paroxysm of tachycardia the ectopic focus, wherever it may be, ceases suddenly to produce contractions and there is a pause, which is usually longer than the interval between beats of the normal slow rhythm—sometimes as long as several of these intervals. The sinus node then begins to function and the normal rhythm is resumed. It can be seen that this is somewhat analogous to the post-extrasystolic pause when the normal sinus rhythm has been interrupted by a premature auricular beat.

AURICULAR FLUTTER

Paroxysmal attacks of rapid heart action may occur through a still different mechanism called auricular flutter. At times auricular flutter may continue for weeks or months. It is shown in Figure 38 A, B and C. The typical feature is the continual up-and-down wavy movement of the base line at a rate of about 300 per minute, each peak being about 1/5 sec. from the next. This wavy line is due to the auricular activity and has the ventricular waves superimposed upon

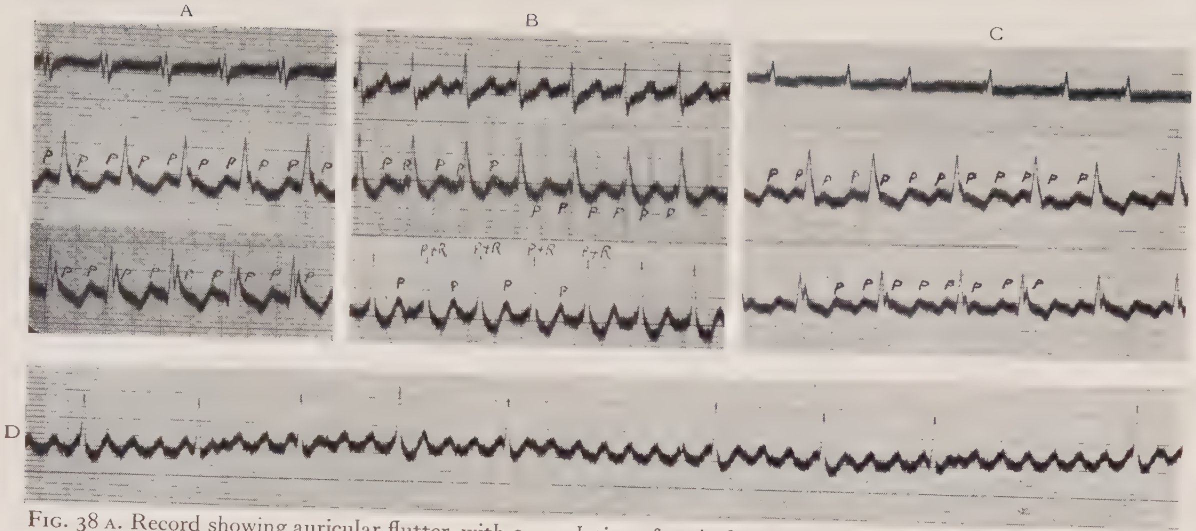

Fig. 38 a. Record showing auricular flutter, with 2 : 1 relation of auricular and ventricular waves. Auricular rate 288 per minute.

b. Another record of auricular flutter showing 2 : 1 relation of auricles and ventricles. Auricular rate 294 per minute.

c. Later record from the patient who gave a, showing a 2 : 1 or 3 : 1 auriculoventricular relation. Auricular rate 288 per minute. The auricular waves are smaller in this record. The patient was somewhat under the influence of digitalis.

d. Record by Lead 3 from the patient who gave Record b, to show the result of pressure upon the vagus nerve in the neck. This record was taken immediately after Record b.

it. It is usually best seen in Leads 2 and 3. In Lead 1 the waves due to the auricles may be but slight. It is difficult to say which is the zero level of these records. All records of auricular flutter show this practically continuous movement of the base line, but with the slower rates, i.e., below 255, it seems as if the zero level were midway between the peak and the trough of these waves, the P deflection being first sharply downward and then rising rather sharply above zero, to return to it again before the next wave starts. This can be observed in Figure 38 C, and less plainly in Figure 38 A. Records which have caught the onset or cessation of auricular flutter bear out this statement, for the zero level of the record during normal rhythm lies midway between the peak and trough of the wavy movement due to the flutter. It is probable however that different patients may have waves of different form with auricular flutter, depending upon the special path of the contraction in the auricular muscle.

The auricles appear to have practically no diastolic period, one electrical wave continuing on into the next; but it must be that some part of the potential of these waves is produced during the diastole of an appreciable part of the auricular wall, for the polygraph shows a succession of pressure waves due to successive auricular contractions and relaxations. The fall of the polygraph waves is due to a fall of intra-auricular pressure, and must mean diastole of at least the major part of the muscle of the auricle. Whatever causes the electrical deflection during diastole must involve the activity of only a small part of the auricular muscle.

The rate of the auricular waves varies in different records from about 240 to as much as 310 per minute. In Figure 38 it is 288 per minute for Record A and 294 for B and C. The ventricles respond to every second auricular impulse in Records A and B, and irregularly to the second or third impulse in Record C.

Figure 38 D shows the result of vagus pressure upon the A-V conduction in the patient who gave Record B. Pressure upon the vagus nerve in the neck on either right or left

side *never fails to slow the ventricular rate* when flutter is present. Figure 38 D was obtained immediately after 38 B was recorded, but during pressure on the left vagus. The effect of vagus pressure on the P waves is very slight and transient, consisting of a very slight change in the rate. This change usually lasts only a second or two, but is perfectly measurable in my records and in all the records so far published in medical literature showing the effect of vagus pressure in the presence of flutter. It can be seen in Figure 38 D. It is a variable effect, sometimes slowing and sometimes quickening the rate by a few beats per minute. Lewis has noted a similar variable effect upon the auricular rate of flutter when the patient changes from the standing to the lying position.

A 2:1 relation of auricular and ventricular waves is the usual thing with auricular flutter, but this depends upon the functional condition of the A-V bundle, and 3:1 or 4:1 or irregular ventricular responses are commonly found. Cases showing 1:1 response have been reported and the records published. The rate has usually been slower than 250 per minute. The interpretation of polygraphic records is too unreliable to be depended upon when the rate is as rapid as this; even with the electrocardiogram it is difficult.

The 2:1 relation of auricular rate to ventricular is not evidence of subnormal function of the A-V bundle with these rapid rates, for the impulse from the second auricular beat arrives while the ventricle is still contracting in response to the impulse from the first auricular beat, so that it finds the ventricle refractory to stimulation. If two or more auricular beats fail, however, one of the impulses must have been blocked in the A-V system. The blocking shown in Figure 38 C was due to digitalis.

The *physiology* of auricular flutter is fundamentally quite different from that of auricular tachycardia. It seems that there is no relation between these two rhythms, flutter being a more marked disturbance of auricular function.

There is one curious and hitherto unnoted feature in their dissimilarity. The rate of auricular tachycardia is almost always less than 200, or at most, 215 per minute, and the electrocardiographic curves of auricular flutter show, with but rare exceptions, an auricular rate of over 240 per minute.

Tachycardia tends to have lower auricular rates, rarely above 215 per minute, while auricular flutter tends to higher ones, usually above 240 per minute. The strong similarity between all curves of auricular flutter, as contrasted with the variable appearance of the auricular waves in tachycardia, is additional evidence for a physiologic difference between these two mechanisms, and in favor of a common mechanism for all cases of flutter.

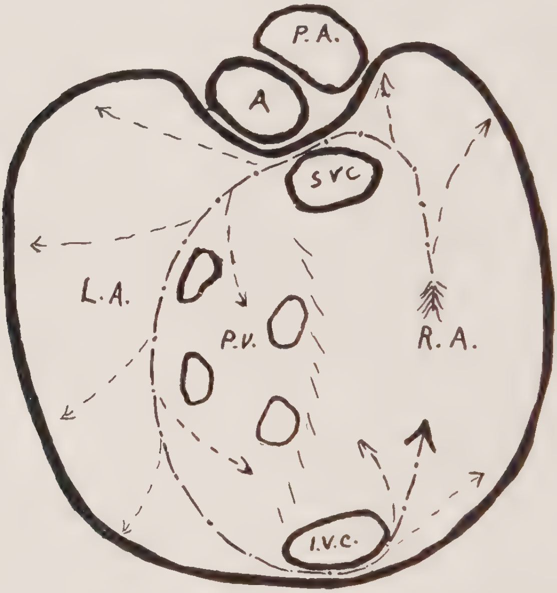

Fig. 39. Diagrammatic view of the auricles from behind, to indicate the usual course of the circus contraction of auricular flutter. The central circus is seen to surround the superior vena cava (S. V. C.) pulmonary veins (P. V.) and inferior vena cava (I. V. C.). There is a gap of quiescent muscle between the tail and the head of the contracting portion. The contraction progresses in the direction indicated and spreads to the remainder of the auricular muscle, as shown by the arrows radiating from this central path.

Lewis has put forward an hypothesis as to the mechanism of auricular flutter based upon the observations of Mines and Garrey and elaborated by experiments of his own. It is in such excellent agreement with all that had been brought forward previously, that it seems undoubtedly correct. Flutter, he believes, is brought about by a change in the physiological condition of the muscle, that *delays the rate at which the contraction passes through it.* Thus the contraction wave, having swept across a part of the wall of the auricle, and returned to its point of origin, finds the contraction process completely passed away there, so that the region is again capable of contracting. This it does, and the contraction spreads again along its former path, and again on returning to the starting point this has ceased to be refractory, is again irritable, and again contracts. There is thus formed a ring, around which the contraction wave is coursing. Behind the contracting portion of the ring is an area which is relaxed and which enters into contraction as the front of the wave proceeds (Fig. 39).

This process was described as a *circulating rhythm* by Mines, who said: "Such a wave (of contraction) runs round the ring (of auricular muscle of the ray) sufficiently slowly for the refractory phase to have passed off in each part of the ring when the wave approaches it. Thus the wave circulates and may continue to do so." Garrey introduced the term "circus contraction," which is so descriptive that it seems worth retaining.

In most of the dogs which he investigated, Lewis found that the path of the "circus contraction" was around the entrance into the auricles of the superior vena cava, right pulmonary veins and inferior vena cava, as shown in Figure 39, which is modified from one of his illustrations. In others it surrounded only the superior cava and the right pulmonary veins, and in one dog it appeared to surround the left A-V orifice. From the central path of the "circus contraction," the other parts of the auricles are involved by a radial spreading of the contraction as shown in the figure, and with each "circus contraction" this radial spreading is repeated. The radial paths, like the central circus, are always the same, and thus the auricular waves are always the same.

AURICULAR FIBRILLATION

Paroxysmal attacks of rapid ventricular rate may also be caused by the sudden inception of what is called fibrillation of the auricles. But fibrillation may also be a permanent condition, and in fact, usually is so. In either case the ventricles beat irregularly. Electrocardiograms of this condition are shown in Figure 40, the typical characteristics being: (1) absence of a wave of constant form which might be due to auricular contraction preceding each ventricular wave; (2) in the interval between the ventricular complexes a series of wavelets marked f f f which vary in height and width (i.e., rate). Certain of these waves may closely resemble a P wave and may occur at the proper interval before some of the ventricular complexes, but if the record is examined at large it will be found that at best this is only an occasional occurrence, as others of these waves will have slightly different form or will occur at a different interval before the ventricular complex.

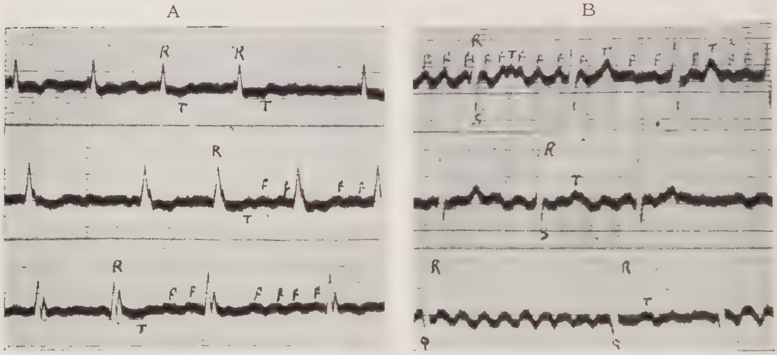

FIG. 40 A. Auricular fibrillation. The small wavelets marked f f f are due to the auricular activity. The ventricular waves occur irregularly and are not preceded by any wave of constant form which might be a P wave. Note the slurring or notching of the Q R S group in three leads.

B. Auricular fibrillation to show how large the auricular waves may sometimes appear. The ventricular complexes show right axis deviation of Q R S.

Figure 40 A is from the patient who had previously given the records A and C of Figure 38, and it is seen that the

regular waves of auricular flutter have given way to a quicker series of waves which are not so large and which diminish from time to time until there is almost no movement of the base line. In different patients, the rate of these waves varies from 350 to 500 per minute, but the characteristic feature is that they are constantly changing form, and that they tend alternately to increase and decrease in size. Figure 40 B shows a record with extraordinarily plain auricular waves which might suggest a diagnosis of auricular flutter.

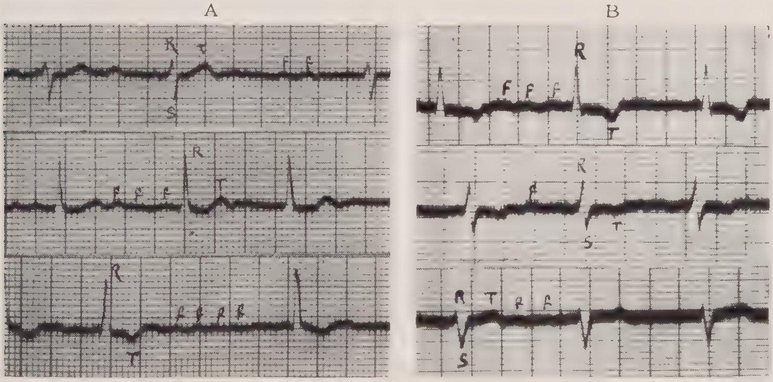

FIG. 41 A. Auricular fibrillation with small excursions of the auricular waves. The ventricular complexes show right axis deviation of Q R S.
B. Auricular fibrillation to show very small auricular excursions. The ventricular complexes show left axis deviation of Q R S and a downward T wave in Leads 1 and 2.

These waves appear to be regular in parts of the record, but if carefully measured are found not to be so. Moreover they vary in *height* and *form*, and in parts of the record can be seen to disappear almost entirely. This is in strong contrast to auricular flutter, whose waves measure equally over long intervals and are of uniform height and form. A more typical record of auricular fibrillation is seen in Figure 40 A. These last two cases have a rate of about 400 per minute for the slowest fibrillation waves. Other records may show rates as high as 500 per minute.

The height of the waves due to auricular fibrillation seems to depend upon the same combination of factors as does the height of P (p. 59). At least they are found large most often with hearts that have mitral stenosis (auricular hypertrophy), and small most often with hearts that have diffuse myocardial disease and are in poor condition. Figure 41 B is from a case of diffuse disease and the record shows only small fibrillation waves, varying in size and sometimes disappearing altogether for long stretches.

During fibrillation of the auricles there is no coordinate contraction of the muscle fibers of these chambers sufficient to cause a pressure variation within them. This fact was apparent in polygraphic records and led to an hypothesis of auricular paralysis. Individual fibers or groups of fibers continue to contract, but the shortening of some fibers is neutralized by the synchronous relaxation of others, so that the net result is a practically constant size of the auricular wall.

Lewis has pointed out that there is a relation between the auricular physiology in flutter and in fibrillation. In each case there is a *circus contraction* but in fibrillation its path is not always the same. There is an increase in the rate of movement (propagation) of the contraction in the central ring, and a shortening of the duration of the contraction (refractory period) of the individual muscle fibers. This leads to continual variations in the length and course of the central path, and therefore to variations in the radial paths by which the contraction spreads from the central ring to the outlying parts of the auricular muscle. This theory should, I believe, be accepted. It goes far to make clear many of the clinical and experimental features of auricular fibrillation.

Several authors have observed in experimental animals what they considered to be a combination of auricular flutter and auricular fibrillation. In addition to the "rapid series of contraction waves passing over the auricular wall" which can be plainly seen when the auricles are in flutter, there were "simultaneous fine fibrillary movements, more especially observed along the auriculoventricular groove and in the appendices." It may be that this condition is responsible for the large well-defined fibrillation waves of such records as Figure 40 B and 42 A which strongly suggest the wavy line of flutter. These records may be referred to as showing coarse fibrillation waves, but it is clear that they indicate fibrillation and not flutter.

We must distinguish from this a condition, obtained in experiment, that Lewis calls "impure flutter." This is characterized by slight irregularity in the form and duration of certain individual flutter waves, while nevertheless the predominant auricular rhythm of flutter is maintained. This may be disturbed temporarily by the abnormally shaped waves but is not destroyed. In the same way a normal auricular rhythm may be disturbed by premature beats of various sorts, but nevertheless continues dominant. I have seen but few clinical records which entirely fulfill these qualifications. One patient eventually showed typical auricular fibrillation; the others could not be followed.

The *ventricular response during auricular fibrillation* depends upon the number of effective impulses which are passed to the ventricles by the A-V system. The integrity of the function of this system is a very important factor in determining the ventricular rate, though it has never been proved that there may not be another factor. There may be a qualitative difference in different auricular stimuli giving rise to the A-V impulses and only certain stimuli are effective. It may be that impulses come to the A-V node irregularly, or there may be a summation of small stimuli in the node until the level of impulse formation is reached.

The usual thing when auricular fibrillation sets in is to find the ventricular rate, as in Figure 42 A, very close to the maximum which could be attained, considering the fact that the ventricles are refractory to a second stimulus until the T wave is completed. In this record the rate is 186 per minute and the irregularity very slight. It would scarcely be appreciated by the finger on the pulse and can be recognized only by very careful auscultation at the apex. Pressure upon the vagus nerve in the neck always produces a slowing of the ventricular rate and makes the irregularity more

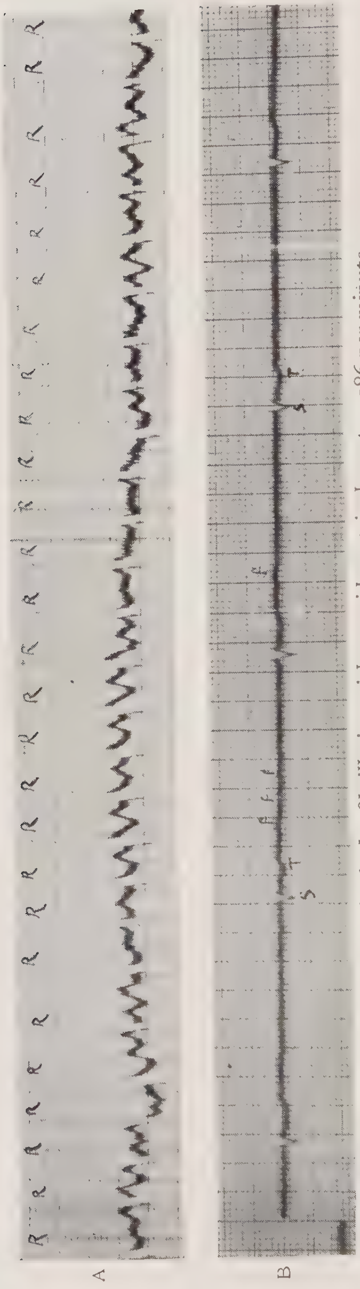

Fig. 42 A. Auricular fibrillation with a rapid ventricular rate—186 per minute.

B. Auricular fibrillation combined with complete heart-block. The ventricles are perfectly regular at 36 per minute. Both the auricular and ventricular waves were as small in the other leads as in this one.

evident by blocking some of the impulses from the auricles. Digitalis slows the ventricular rate, by its ability to increase the activity of the vagus, and disease of the node or main stem of the bundle also reduces this rate.

When the rate is below 120 per minute the irregularity is more evident than at higher rates, but when it is 75 or 70 or less, irregularity again becomes less apparent. Some patients with fibrillation of the auricles continually maintain a ventricular rate of 75 or under without taking any medication. Such patients should be considered to have either a vagotonia or an impairment of the A-V system due to disease. Without this they would have a ventricular rate of 90 to 100 or more, which is the lowest rate that a patient unaided by digitalis seems able to attain through the normal activity of the vagus.

When auricular fibrillation is present and the ventricles are slow and perfectly regular, as shown in Figure 42 B, we know that complete heart-block is present. This is true, no matter if the ventricles should be faster, for if the A-V system is capable of functioning at all it will transmit impulses irregularly when the auricles are fibrillating.

Aberrant ventricular complexes, like those found with auricular premature beats, are especially common with auricular fibrillation when the rate is rapid, as are also premature beats of ventricular origin.

Ventricular Fibrillation

Just as the auricular muscle goes into fibrillation, so the ventricular muscle may take on this same incoordinate activity under certain circumstances. It is probable that ventricular fibrillation occurs much more frequently than is observed, for if it lasts more than a few seconds the patient cannot live. If the ventricles do not beat coordinately they will cease to drive the blood and the patient will die. It is probable that the inception of ventricular fibrillation is the cause of death in most patients who die from embolism or thrombosis of a main coronary artery or of a large branch, and in many patients who have extreme dilatation of the ventricles due to cardiac failure. Either of these conditions can cause ventricular fibrillation in animal experiments and so probably in human beings.

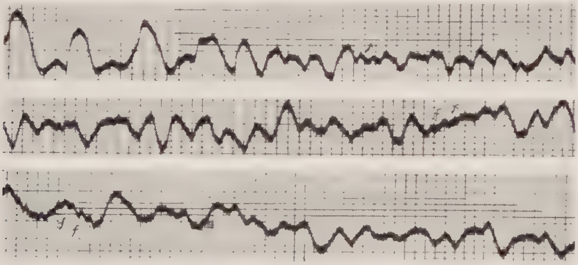

Fig. 43. Ventricular fibrillation. The same sort of irregularly varying form is seen in this record as in the auricular waves of fibrillation of the auricles, but the waves are much larger and often bear a certain resemblance to ventricular extrasystoles. The small wavelets ff are considered due to auricular fibrillation. This illustration is from Dr. Halsey's article on ventricular fibrillation.

Figure 43 is a record showing fibrillation of the human ventricle, and is seen to bear some resemblance to the irregular waves due to auricular fibrillation, though the waves are much larger. There are parts of the record which suggest deformed premature ventricular beats, but in other parts are waves of 0.4 sec. duration which are probably due to the fibrillary contractions of the ventricular fibers. The deformed ectopic beats probably correspond for the ventricles to what the deformed flutter waves mean for the auricles when they are in fibrillation. Records have been published which showed a period of ventricular fibrillation followed by recovery of the normal rhythm, but it is likely that the most frequent occurrence is for it to end in cardiac stoppage and death.

Ventricular fibrillation occurs experimentally in hearts which are subject to different sorts of poisoning. The causes are as various as lobar pneumonia and chloroform plus asphyxia. It also occurs in severely failing hearts and, as has been said, after coronary artery occlusion. Tying off a main coronary artery in a dog practically always leads to a period of ventricular tachycardia which passes into fibrillation of the ventricles. It will be rarely recognized clinically, for it must, from its result upon the circulation, be of very brief duration.

CHAPTER VII

CLINICAL ASPECTS OF DISTURBANCES IN RATE OR RHYTHM

When the mechanism that produces the orderly sequence of the heartbeat is disturbed, irregularity of the ventricular systoles will usually result. There are, however, quite similar disturbances of the normal heartbeat that do not cause irregularity. The ventricular systoles may be rapid and regular, or slow and regular, or the rate may even be regular and within normal limits with an abnormal mechanism in effect. We shall consider the clinical aspect of all the different abnormalities of cardiac rhythm together, because of the likeness of their mechanism, even though the result may sometimes be a regular beat and at other times an irregular one. The arrhythmia is not so important as the fact that the heartbeat has an abnormal origin.

When considering disturbances in rate and rhythm of the heart it will be necessary to keep in mind the relation of the auricular systole to that of the ventricles, and also, because the pulse is so often carelessly taken to indicate the heart action, the relation of the ventricular systole to the occurrence of a pulse wave. Under normal conditions each auricular systole is followed by a ventricular systole and this is followed by a pulse wave which passes to the periphery. If the auricles for some reason should omit one beat the ventricles would also omit this beat and there would be one pulse beat missing. If the auricles should stop for a long period the ventricles would also stop and there would be no pulse, until after a time the ventricles would become roused to an activity of their own (ventricular escape) and would begin to beat, usually at a regular rate of about 30 beats per minute. As far as is known, auricular stoppage is always followed after an interval by spontaneous ventricular activity, unless the auricles should begin first, in which case the normal succession of the heartbeat is again carried on.

When the heart is irregular the size of each pulse wave is dependent upon the length of the pause preceding it. Variations in the length of diastole will cause variations in the degree of filling of the ventricles. If the pause is long the ventricles will be well filled and the pulse wave will be large; if short, there will be but little blood to be ejected into the aorta by the contraction, and there may be only a small pulse or perhaps none at all. Pulse rate or rhythm should never be taken to indicate heart rate or rhythm. The physician should always listen over the apex of the heart to be certain of the regularity or irregularity of the heart action.

The electrocardiogram has contributed greatly to the understanding of abnormal cardiac mechanisms, so that now we are able to diagnose them correctly in the majority of instances without a record being taken. There are times, however, when a record will show our clinical diagnosis to be incorrect, and there are certain conditions, such as prolonged auriculoventricular conduction time, which would never be suspected without instrumental aid.

The polygraph can also give information as to the cardiac mechanism, and it has the advantage of greater portability. If an electrocardiographic record can be obtained there are definite advantages in its favor. A good readable record can be obtained with greater ease and certainty, the reading of the waves is more definite, and moreover, beyond the mere rhythm changes, the information which the electrocardiogram can give regarding the state of the ventricular muscle (Chap. V) is not even hinted at by polygraphic records.

Sinus Arrhythmia

Sinus arrhythmia is extremely common. A slight degree of arrhythmia can be found on measuring the records of almost every heart whose rate is under 80 per minute. This would be almost or quite imperceptible to one listening at the apex beat or feeling the radial pulse. A degree of sinus arrhythmia which can be observed by these latter methods is also quite common, especially in those under thirty and over fifty years of age, and in any person after defervescence from an acute infection. It is noted in practically everyone when the heart slows after exercise has caused it to accelerate.

The clinical significance of finding a sinus arrhythmia is only that of overactivity of the vagus nerve, even though the arrhythmia may have caused symptoms referred to the heart or circulatory system. This arrhythmia is usually symptomless, but the more extreme types, such as sinoauricular block, sudden temporary heart stoppage, or periods of very slow heart, are likely to cause dizzy spells, palpitation or fainting. No matter how severe these symptoms may appear, they should never be cause for alarm. The disease, if any, is always extracardiac and the symptoms are due to a temporary functional depression of the heart rate with the resulting decreased output of blood.

Premature Beats

The clinical significance of premature beats is very variable from one case to another, for it depends partly upon their cause and partly upon whether their origin is in the auricles, the ventricles or the junctional tissues. A premature beat may always be viewed as an expression of a hyperirritable focus in the heart.

In experimental animals it is possible to produce premature beats from the normal auricles or ventricles by electrical stimulation of the sympathic nerves, by the combined action of chloroform and asphyxia and by the use of various drugs. The most important of these because of their use in medicine are morphin, strophanthin, digitalis, adrenalin and caffeine.

The human heart may also give rise to physiological premature beats which may arise in any part of the heart. They are frequently found as a result of Graves' disease, and are probably then due to the overactivity of the autonomic nervous system which accompanies this condition. They occur also in neurotic individuals and probably then result from the generally exalted nervous activity.

Premature beats occur commonly with organic disease of the gall-bladder and less commonly with disease of the stomach, intestines, kidney or lungs. The mechanism here is probably a reflex through the autonomic and sympathetic systems.

Premature beats may also result from the action of drugs, digitalis, morphin, coffee, tea and tobacco being of most clinical importance. Stopping the drug will of course abolish the arrhythmia if it should be the cause.

In spite of these not uncommon extracardiac causes of premature beats it is true that many patients with this irregularity have as a cause a pathological condition in the heart. The diseased area need not be a large one and its seriousness for the heart as a whole is often slight. A small focus of streptococcic or syphilitic infection or a small area of deficient blood supply due to coronary atheroma is capable of causing this disturbance and might not affect the heart's function appreciably.

In a series of 121 cases with premature beats studied by Lewis, 71 per cent of the patients were affected by one or another definite form of disease of the heart, and the other 29 per cent could be divided as follows: 19 per cent had active disease elsewhere in the body, such as bronchitis, lumbago, gastric disease etc. (2 cases of exophthalmic goiter are included here), and 10 per cent, except for the irregularity, were apparently healthy. In a series of 50 cases which I have reviewed from this standpoint the proportions in these groups are 84 per cent with cardiac disease, 10 per cent with active disease elsewhere and 6 per cent without any disease being found. The difference between these two sets of figures probably arises from differences in the types of patients included in the two series. As an example of the effect which this sort of selection may have I may cite Mackenzie's statement that 50 per cent of women show premature beats during pregnancy. Supposing a study of the conditions associated with premature beats were made in an obstetrical clinic. The figures would show a larger percentage than Lewis' of cases in which no disease could be demonstrated. As both Lewis' and the author's series are taken from groups of patients who predominantly have more or less serious cardiac disease it is only surprising that so many with the irregularity did not also have evident disease.

In many of those in whom no disease is found the irregularity is due to reflex or other nervous causes. It seems likely, though, when we consider how insidious must be the onset of arteriosclerotic heart disease, that patients over forty or forty-five years of age might have premature beats as the only sign of a small localized area of myocardial degeneration.

The frequency of occurrence of the premature beats has a bearing on their prognosis. A premature beat entails a certain amount of wasted effort on the part of the heart, for it contracts upon a ventricle which, because of the short preceding diastole is only poorly filled with blood. There can be but a small output of blood from this beat at best, and there is often none at all, yet the cardiac contraction expends as much energy as if the ventricle were full. When the premature beat occurs infrequently it does not amount to a serious loss of cardiac power, but the more frequently it occurs, the more of the heart's energy is wasted. It has been shown that in the dog, artificially produced premature beats occurring 4 to 8 times per minute change the blood flow but little, the greatest loss being 11 per cent; and some experiments show no change worthy of note. When the premature beats come more often they cause a considerable falling off in the blood flow.

From quite another point of view a greater frequency of occurrence is an undesirable prognostic factor. Frequently occurring premature beats are likely to be caused by a disease of the myocardium rather than by nervous influences, while the more infrequent ones are usually due to nervous influences. This statement is not without special exceptions in both of its phases; especially when the premature beats occur regularly after every other normal beat. These are usually of a benign origin. When two or three premature beats occur in succession, the cause, in my experience, has always been myocardial.

The circumstances under which the premature beats occur also give a clue to their importance. When they appear after digitalis administration they have no significance other than that the drug is causing what has been called one of its minor toxic phenomena. When tobacco, tea, coffee or alcohol are used to excess, the premature beats may be due to these drugs, and may disappear when they are stopped.

Premature beats sometimes are present only when the patient is quiet and the heart rate slow, and disappear when the rate becomes more rapid, from whatever cause. In such cases the premature beats are especially likely to occur when the heart slows down just after a period of exercise, probably because of the activity of the vagus and sympathetic system at this time. These are usually benign cases without a serious foundation.

In other patients the premature beats will become more frequent when the heart is accelerated by exercise, and these are usually due to a myocardial focus. Likewise when premature beats occur in the course of an acute infection, such as scarlet fever, rheumatic fever or pneumonia, they are considered to indicate an invasion of the heart muscle by the disease.

The prognosis quite evidently does not depend upon the presence of the premature beats so much as upon their cause. We shall feel that they are due to heart disease if such other signs are present as the murmurs of valvular disease, an abnormal electrocardiogram of the rhythmic beats, a decrease of the cardiac reserve power, sufficient cardiac enlargement to be unquestionable, or an increase in the irregularity during the twenty seconds immediately after exercise. If none of these things are observed we must conclude that the arrhythmia is not due to muscle disease of any extent or severity. The next step is to look for the reflex or nervous factors which have been mentioned as possible causes of the arrhythmia. If none of these are found we may feel that it is without clinical importance.

It makes some difference whether the disease focus is in the auricles or the ventricles. The sequel of auricular myocarditis is fibrillation of the auricles, while that of ventricular myocarditis is cardiac failure. This difference is not a great one, for it is an uncommon thing that the auricles and ventricles are affected by disease to a very different degree. The sequel of myocarditis in the auriculoventricular node is heart-block; but disease is not usually sharply localized in this situation, either, for auricles or ventricles or both are likely to be involved at the same time.

The outlook of the patient with premature beats is plainly not affected by the mere irregularity. It depends upon a careful decision as to the cause of the irregularity, the possibility of removing the cause and a careful consideration of the effect upon the heart's efficiency if the causal condition should persist.

Heart-block

The recognition of the higher grades of heart-block is usually not difficult even without instrumental aid, because of the slowness of the heart rate and the characteristic response by further slowing to pressure stimulation of the vagus in the neck. This will not, of course, be obtained when complete block is present, but the rate then is usually so slow as to be characteristic (30 per minute or less.) The lesser grades of block with dropped beats may be confused with premature contractions. The pulse may be bigeminal or trigeminal, just as when premature beats occur regularly. One can usually distinguish which mechanism is present even without a record, by listening carefully at the apex of the heart for the sounds of a premature beat. Pressure on the vagus in the neck will help our decision, for it will increase the frequency of dropped beats due to heart-block, though it will not increase the frequency of premature beats.

The lowest grade of block with prolonged conduction time between auricles and ventricles cannot be determined without the polygraph or the electrocardiograph. Prolonged conduction time, perhaps with occasional dropped beats, may be the only evidence of cardiac involvement appearing during the course of a rheumatic infection, pneumonia, diphtheria or other acute disease. It is common in these infections for the disease of node or bundle to be part of a more or less wide-spread myocarditis. Such acute processes increase the gravity of the outlook; but should the patient survive, they almost always resolve to such an extent that the conduction time becomes normal again, and the heart shows no sign of having been affected. The persistent heart-block which is common clinically may be due to repetitions of these minor insults, but is usually a chronic destructive process involving the node or bundle. The cause is sometimes rheumatic, sometimes syphilitic, but usually arteriosclerotic. With this etiology we can scarcely hope for a recovery of the conducting function; so that the condition of the patient, as well as his outlook for the future, will depend upon the integrity of his ventricular muscle, how much it is affected by the disease, and how well it is able to compensate, by an increased strength of the individual beat, for the decreased frequency of the contractions.

In partial block the immediate outlook is governed but slightly by the number of dropped beats per minute, though a rate under 50 per minute is a definite mechanical disadvantage. The quality of the ventricular muscle, its relative freedom from disease, is always the predominant factor in the prognosis, and for this reason the ventricular complexes should be examined for evidences of myocardial abnormality.

TACHYCARDIA

There is a very distinct clinical separation between the tachycardia which we have termed physiological and that which is paroxysmal and might be called pathological. The physiological tachycardia is the response of the heart to nervous influences, to some toxin in the blood, or to a condition of undernourishment of its muscle. It occurs with fever, hyperthyroidism, excitement, anemia, cardiac failure etc. The rapid heart which occurs with intracranial conditions such as apoplexy, is probably due to a depression of the vagus center releasing the heart from its normal retarding influences. Physiological tachycardias do not necessarily mean disease of the heart, and are never the principal cause of heart failure, though they may be a contributing cause by leading to cardiac fatigue. Long continued rapid heart action will lead to undernourishment of the heart muscle and consequently to cardiac fatigue and failure, but usually the toxemia which causes the rapid rate damages these hearts more than the rate itself. Paroxysmal tachycardia is often due to disease and often not. This is of course a distinction of importance and is determined along such general lines as have been detailed for premature beats (p. 144).

The clinical significance of paroxysmal tachycardia differs according to the part of the heart in which it originates. Paroxysms arising in the auricles and in the auriculo-ventricular node are more often due to a disturbance of the cardiac nervous mechanism than to disease. It is certain that in young people they often cease to occur after a time, and the heart seems to remain intact.

Ventricular tachycardia does not occur except from cardiac disease. It is especially liable to be caused by disease of the coronary arteries.

The clinical importance depends too upon the readiness with which the attacks can be stopped. Auricular and nodal foci are more under the control of the vagus nerve than are ventricular foci, and vagus pressure or other forms of vagus stimulation, including drugs which have this action, are more likely to stop attacks originating from these foci than those originating in the ventricles. The prognosis of tachycardia of auricular or nodal origin is therefore better in this respect also.

Auricular Flutter

Auricular flutter comes on in sudden attacks, as does paroxysmal tachycardia, and these attacks may end as suddenly. Without an electrocardiogram it cannot be certainly diagnosed except after several observations. Auricular flutter will sometimes occur in a chronic form persisting for months or years, or it may give place to auricular fibrillation. The auricular rate is usually between 260 and 320 per minute, and the usual 2:1 A-V ratio makes a ventricular rate close to 150, the usual finding. In long-continued cases the blocking is usually greater, so that the ventricles are slower and perhaps irregular. In the latter case the condition simulates very closely indeed the rhythm of auricular fibrillation. The reaction to vagus pressure by a slowed and irregular ventricular response is a characteristic feature of flutter, but this change lasts only during the vagus pressure, and the rapid regular rate promptly returns. Digitalis administration will slow the ventricular rate as promptly as the patient's therapeutic dosage is reached, and all patients with this condition should have the rate properly controlled by digitalis medication. Under digitalis the rate usually becomes irregular as well as slower.

Auricular flutter is probably always dependent upon a pathological process. Careful microscopic studies of the auricular muscle in this condition are not numerous, but they usually show a diffuse fibrosis, perhaps due to chronic arterial disease or to a diffuse leucocytic infiltration. It occurs in chronic arteriosclerotic patients and in others who have an interstitial myocarditis, whether acute or chronic. It occurs during acute infections, particularly acute rheumatic fever, lobar pneumonia and diphtheria, and after syphilitic infection. A patient having an attack during an acute infection may recover from it and subsequently have no signs of cardiac abnormality. On the other hand, those who have auricular flutter as the result of chronic processes usually have some limitation of their cardiac reserve even in the intervals between attacks. This shows that the ventricles as well as the auricles are affected by the myocardial disease.

Patients who have had one attack, unless it occurred during an acute infection, are very likely to have another. They are also likely to have auricular fibrillation, either coming on suddenly with an attack of rapid irregular ventricular action, or as a sequel to a period of flutter. The physiological relation between flutter and fibrillation of the auricles appears to be extremely close, certain cases changing repeatedly from one mechanism to the other during periods of rapid ventricular rate.

The rapid ventricular action due to auricular flutter can be readily controlled by digitalis. This drug also affects the auricular activity, frequently changing flutter into fibrillation. If digitalis now be stopped the normal rhythm will sometimes return, but fibrillation may continue or may revert again to flutter, so that we may be continually forced to deal with an abnormal cardiac mechanism. Quinidine given directly the condition is recognized is usually successful in causing a reversion to normal rhythm, though there are some cases not so affected.

If the ventricular rate is properly controlled by digitalis the patient can go about and do a great deal while auricular flutter is present. The situation in this respect is quite similar to that with auricular fibrillation. The ultimate outlook for these patients depends more upon the degree of valvular disease or hypertension, the integrity of the ventricular muscle and the proper control of the ventricular rate by digitalis than upon the character of the auricular activity. The prognosis for the attack, with the use of proper treatment, is good, but the ultimate outlook is to be determined by the extent of the cardiac disease, on such grounds as are discussed under auricular fibrillation.

Auricular Fibrillation

Clinically, auricular fibrillation appears in a paroxysmal and also in a chronic form. The chronic cases often give a history of one or more paroxysmal attacks, one of which finally persists. As has been mentioned, auricular fibrillation may be a sequel to auricular flutter. When this occurs the fibrillation may either revert to flutter, give place to normal rhythm or remain persistent.

The paroxysmal form of auricular fibrillation, as it appears clinically, differs from paroxysmal attacks of auricular flutter only in the irregularity of the ventricles. The symptoms, treatment and prognosis are practically the same. When auricular fibrillation is long-established it is usually permanent, though I have seen long-standing cases revert for a time to normal rhythm, and such cases are found in the literature. Quinidine, when properly given, will frequently cause even long-standing auricular fibrillation to give place to normal rhythm, and this will often remain for a considerable period, though it sometimes does not. If the fibrillation returns, quinidine may again cause reversion to normal rhythm. Thus for many patients the onset of permanent fibrillation can be considerably postponed.

When fibrillation of the auricles is permanently established the prognosis for each individual case demands, above all else, that the proper amount of digitalis be used to slow the ventricular rate and keep it continually between 70 and 80 per minute while the patient is at rest. Without such medication the tendency is almost universally to heart failure through exhaustion of the heart muscle by the rapid ventricular beating. With occasional patients who have a pathological lesion in the A-V system or a vagotonia, so that some of the impulses from the auricles are prevented from reaching the ventricles, the rate does not tend to be rapid even without digitalis.

The prognosis depends, first of all, upon a proper treatment with digitalis, for even the best heart will fail without it. Granting this, the prognosis depends, as in all the other arrhythmias, upon the degree of involvement of the ventricular muscle by the disease which has caused the arrhythmia, and upon the mechanical handicap, such as valvular lesions or high blood-pressure, against which the muscle must work.

Auricular fibrillation is probably always a result of myocarditis within the auricles. It may be caused experimentally by the injection of certain drugs which damage the heart muscle, and clinically may occur from acute toxic degeneration due to disease or to infection within the heart itself. The temporary development of auricular fibrillation has been reported as a feature of hydrogen sulphide poisoning. The toxic action of this chemical upon the auricular muscle was the probable cause of the disturbance.

The chronic form is usually due to a chronic fibrotic process within the auricular muscle. This may be residual from a former acute condition, or may be due to arteriosclerosis. Disease of the mitral valve is especially often associated with auricular fibrillation, about 50 per cent of all cases of auricular fibrillation having an associated mitral disease, and about 30 per cent having chronic myocardial, nephritic or arterial disease. There are, however, a certain number of hearts with auricular fibrillation, whose auricles reveal, under the microscope, comparatively little disease. Probably in these, and in all paroxysmal cases, abnormal nervous impulses are an important factor in precipitating the onset, and the fibrillation once started tends to perpetuate itself. Cases with slight pathology are possibly those that revert spontaneously to normal rhythm, or do so easily under quinidine. Cases that do not revert under quinidine are possibly those with more marked structural changes in the auricular muscle.

A pathological process that involves the auricles may involve the ventricles but little. This is the reason why some hearts with auricular fibrillation are able to carry on the circulation so well even under the handicap of valvular disease, high blood-pressure, or continual strenuous bodily exertion, provided that the rate is kept from becoming too rapid for the ventricles to function at their best. If the ventricular complexes are of normal form and the valvular disease or increased blood-pressure not too extreme, the patient's cardiac reserve will often be good enough to allow him to do what he wishes with but slight limitation. If these other factors are unfavorable, the handicap from them will probably result in a certain amount of crippling of the patient's ability. The actual irregularity itself, provided the rate is kept within normal limits by proper treatment, probably handicaps the heart but little.

CHAPTER VIII

THE USE OF THE ELECTROCARDIOGRAPH IN THE EXAMINATION OF CARDIAC PATIENTS

In the previous chapters we have reviewed the characteristics of the normal electrocardiogram, and have pointed out the kind of disease or abnormal function that may cause electrocardiographic abnormalities. We shall here consider what sort of information the records are able to give and in what patients the information from the records is likely to be of importance.

The physician who has little more than heard of the method often feels that the mere taking of an electrocardiogram will tell all that is necessary to know about a patient with symptoms referable to the heart. Such is the mental impression produced by a highly complicated method and highly polished apparatus. At the other extreme are those who know too little and who feel that though there may be scientific interest in the records, yet they have no practical value for a physician who is most concerned with the treatment of his patients. This chapter is written with these two groups in mind, to point out the clinical value of the method, and the manner of its application.

HOW TO READ AN ELECTROCARDIOGRAPHIC RECORD

It is difficult for the beginner to know how best to start the examination of an electrocardiographic record. The most striking feature is usually the Q R S group, but the most important feature may be a slight prolongation of the P-R interval, a thing which may well escape attention. One should therefore use a definite procedure when examining these records, so that no detail will escape notice. The following order is suggested as being both logical and complete:

1. Examination of the test of standardization to determine
   (*a*) whether the jump is exactly 10 mm.
   (*b*) whether there is overshooting at the end of the jump.

2. Determine the P wave in each lead.

3. Determine the rate of the auricular systole by counting the number of complete heart cycles in 6 sec. (which would be 30 of the fifth second divisions) and multiplying by 10.

4. Does P occur regularly or irregularly? If irregularly, have the P waves after the short pauses the same form as the others (sinus arrhythmia) or a different form (premature beats)?

5. Have the P waves constantly an abnormal form (height, inverted, notched, wide)?

6. Are the waves of auricular flutter or auricular fibrillation present instead of normal P waves?

7. Determine the function of auriculoventricular conduction by measuring the P-R interval. Do occasional ventricular beats fail to occur after the proper interval?

8. Do ventricular waves always occur at the proper time interval after P or are some premature? If heart block, auricular flutter or auricular fibrillation are present, determine the rate of ventricular systole.

9. Are the ventricular waves the same throughout each lead? If not, determine the cause for the variation.

10. Does the Q R S group show either right or left axis deviation?

11. Does Q R S constantly show a significant abnormality (notching, abnormal height, abnormal duration)?

12. Does T constantly show a significant abnormality (height, direction, form)?

Like any other single method, the electrocardiographic examination has more value for some purposes than for others. It can:

1. Tell whether the heart beat is governed by normal impulses.

2. Indicate the mechanism of any irregularity that may be present.

3. Give a sign of auricular hypertrophy.

4. Record the auriculoventricular conduction time.

5. Suggest that the right or left ventricle, or neither, is preponderantly hypertrophied.

6. Give an indication of the physiological condition of the ventricular muscle.

7. Show evidence of the action of certain drugs upon the heart (digitalis, quinidine, morphin, etc.).

8. Show certain abnormal features of the waves which indicate disease of the ventricular muscle.

The patient who complains of such symptoms as palpitation or shortness of breath is as justly suspected of having heart disease as is the pale-appearing patient of having a blood disease. If we feel the apex beat we find it is abnormal in one way or another, either strong and heaving, or sharp and brief, or very faint, or perhaps irregular. Our suspicion of heart disease is strengthened by these abnormal findings but we must have more facts upon which to base a proper diagnosis. We determine the size of the heart. If this is found to be increased, the heart is certainly abnormal, though a normal size does not deny the presence of disease. In either case we have an additional fact, which gives us a better understanding of the patient's disease. We must listen for murmurs to determine whether the valves are functioning normally, and to the heart sounds to learn what we can from these, but the *only definite signs of disease of the muscle* are to be obtained from the electrocardiogram. A functional test with exercise will indicate roughly the ability of the heart to carry on the circulation, but it does not indicate the presence or absence of disease of the muscle.

To complete the picture, we must look for extracardiac factors which might delay the circulation and tend to produce the patient's symptoms, taking the systolic and diastolic blood pressure, estimating the condition of the arterial walls, examining the lungs to determine whether they may be unable to aerate the blood properly because of emphysema or fibrosis or encroachment on the pleural space. If irregular action of the heart is present, we may, by aid of our experience of similar irregularities, be able to understand its variety and significance. In other cases we may need a record of the action of the auricles and ventricles which can be studied and carefully measured, in order to determine what was going on within the heart to cause the arrhythmia.

If the mechanism of the heartbeat is abnormal in any of the ways described in Chapter VI, it has a definite bearing on the diagnosis of the cardiac condition which has been fully discussed in Chapter VII. If abnormalities of the ventricular waves are found, it is of much more serious import, for it indicates disease of the ventricular muscle. This is probably of greater importance than the finding of a moderate valvular lesion, and equal to the importance of a marked one. The relation between the different abnormalities of Q R S and T and the extent of ventricular muscle disease has been discussed in Chapter V. It will suffice to say here that one can sometimes tell the extent of the disease from the curve, and sometimes not. If normal ventricular waves are found it is fair to conclude that the patient has a ventricular muscle that is probably normal, or has at most only a slight or a localized disease.

The electrocardiogram has its own special variety of information, namely, the mechanism and character of the contraction of the muscle fibers of the heart. An examination of the heart is as incomplete without the electrocardiogram as it is without auscultation or a determination of the size of the heart. By omitting any one of these methods we may deprive ourselves of information of considerable value in the diagnosis.

Because of the effort necessary to obtain an electrocardiographic record, this method of examination must be omitted more often than the simpler methods. For this very reason the clinician should have clearly in mind the kind of information to be obtained from it. He should know when the information of the electrocardiogram is imperative, and also, when he omits this examination, what is the likelihood that he is missing an important feature of his patient's disease.

The problems which the physician should refer to the electrocardiograph for solution will be perhaps more easily appreciated if presented as cases. Therefore various types of patients with heart disease will be considered, and the likelihood of the method giving important information will be discussed in each case. To group the patients for this presentation prominent clinical features will be used, such as the presence or absence of symptoms referable to the heart (dyspnea, edema, or pain on effort), valvular disease, cardiac enlargement, irregular heart action or congenital abnormalities. As certain of these are present, so are we more or less likely to find disease of the ventricular muscle with an abnormal electrocardiogram.

Clinical Groups

1. *Patients with valvular disease and a regular heart with or without cardiac enlargement.* These patients are likely to have an electrocardiogram indicating preponderant hypertrophy of one or the other ventricle. This finding may be of assistance in the diagnosis of the variety of valvular disease, provided that the evidence from the murmurs alone is not conclusive. The subject has been thoroughly discussed in Chapter III and so the following table will serve as a summary of what has already been said:

Valve lesion	Usual	Unusual	Conditions which, coexisting with the valve lesion in question, would explain the unusual direction of the axis of Q R S
Mitral regurgitation	Slight left axis deviation	1. Normal direction of axis of Q R S	1. Early mitral stenosis 2. Marked pulmonary emphysema or chronic tuberculosis 3. Long narrow chest with vertical heart
		2. Right axis deviation	1. Mitral stenosis 2. Combination of two or more of above factors

Valve lesion	Usual	Unusual	Conditions which, coexisting with the valve lesion in question, would explain the unusual direction of axis of Q R S
Mitral stenosis	Right axis deviation	1. Normal direction of axis of Q R S	1. Early slight stenosis with long-standing regurgitation 2. High blood-pressure 3. High diaphragm with transverse heart 4. Aortic regurgitation
		2. Left axis deviation	1. High blood-pressure 2. Aortic regurgitation 3. Combination of two or more of above factors
Aortic regurgitation	Left axis deviation	1. Normal direction of axis of Q R S	1. Mitral stenosis 2. Marked pulmonary emphysema or chronic tuberculosis 3. Long narrow chest with vertical heart
		2. Right axis deviation	1. Mitral stenosis 2. Combination of two or more of above factors

Combined lesions are a more complicated problem, but it is not impossible to work out the result upon the ventricles of the mechanical influences of the different valve lesions. If the direction of the axis of Q R S does not agree with what would be theoretically expected, then we should carefully seek for the reason of the disagreement. Perhaps, if right axis deviation is present, we may be led to change our interpretation of a diastolic rumble at the apex in the presence of aortic regurgitation, considering it due to mitral stenosis rather than to the Flint mechanism. Perhaps, in a search to explain a left axis deviation we may attach a greater significance to a slightly raised blood-pressure, or find a faint murmur of aortic regurgitation which had previously been missed.

The direction of the axis deviation is not as exact as the table would imply, however, and an explanation of the deviation of the electrical axis of Q R S or lack of such deviation in certain records is impossible. Certain cases apparently afford exceptions to the statements which have been made, but it may be only that we have failed to appreciate some determining factor.

Patients in this group without symptoms of cardiac insufficiency rarely show signs of myocardial disease in the electrocardiogram, but about 10 per cent of those with symptoms will be found to have an abnormality of the ventricular complexes, notching of Q R S, an abnormal inversion of T, or both. As a rule the excursions of the waves are of fairly good size, perhaps because of the hypertrophy which causes the axis deviation. A myocardial abnormality plus the valvular disease constitutes a considerable handicap, and these patients are not so likely to recover their former ability under treatment as are those with only the valvular handicap.

Certain patients of this group will show a prolongation of the auriculoventricular conduction time which would not have been suspected without a polygraphic or electrocardiographic record. This abnormality does not add greatly to the gravity of the prognosis, for the disease does not always progress to the higher grades of block; but if we know of its presence we shall be prepared for the appearance of dropped beats upon thorough digitalization.

2. *Patients who have valvular disease with or without enlargement of the heart and whose heart action is irregular.* If the rate is rapid and symptoms of cardiac failure are present, the irregularity is almost certain to be due to auricular fibrillation. Without the symptoms and rapid rate the arrhythmia might be any of those discussed in Chapter VI. A physician who has made a special study of the characteristic features of the arrhythmias should be able to diagnose the irregularity, after listening to the heart and noting the effect of vagus pressure and exercise. He will probably fail to diagnose the arrhythmia correctly in 8 per cent of cases at the first examination, but later opportunities to study the patient should lower this error to about 5 per cent. Polygraphic records will make a correct diagnosis of the arrhythmia in all but the 2 or 3 per cent of patients from whom a readable record cannot be obtained, namely, those with rapid hearts and a few others who are very adipose.

A decision as to the type of arrhythmia has an important bearing on the probability of myocardial disease, as has been discussed in Chapter VII. The electrocardiogram affords the easiest and plainest diagnosis of the arrhythmia. It will also show by the character of the ventricular complexes whether there is a myocardial process extensive enough to have led to an abnormality in the contraction of the muscle of the ventricles. About 15 per cent of patients with valvular disease and irregular heart action will show abnormal ventricular complexes.

3. *Patients with irregular heart action but without valvular disease.* Without cardiac enlargement or symptoms of cardiac insufficiency, it is rare for the electrocardiograms of these patients to show an abnormality indicating ventricular disease. Perhaps in only 2 or 3 per cent will this occur, and the arrhythmia will then almost always be due to premature beats.

When these patients have cardiac enlargement or symptoms of cardiac insufficiency, or both, the irregularity is usually auricular fibrillation. The record will generally show only a left axis deviation of Q R S of greater or less degree, but perhaps 15 per cent will also have abnormal ventricular complexes indicating muscle disease.

4. *Patients without valvular disease or arrhythmia with symptoms referable to the heart.*

Patients in this group without cardiac enlargement are unlikely to have abnormalities in the electrocardiogram. They comprise for the most part, cases of the irritable heart or neurocirculatory asthenia which was so prominent during the army examinations; some instances of hyperthyroidism will also be included. These patients have no cardiac disease.

If the patient has the retrosternal pain typical of disease of the coronary arteries coming on with exertion and relieved by rest, or has had a severe typical attack of angina pectoris, it is very common to find significant abnormalities of the ventricular waves. About 70 per cent of those patients will show one or more of the abnormalities mentioned even though the heart is quite normal in size and free from murmurs; half of these have the special T-wave peculiarity associated with coronary narrowing.

Of those patients who have cardiac enlargement and symptoms of circulatory insufficiency, the number showing abnormal electrocardiograms will be quite large. These have the diffuse myocardial degeneration from arterial narrowing or from multiple foci of low-grade infection within the muscle, and abnormal ventricular waves are found in about 85 per cent. They are often referred to as having "cardiosclerosis," or as "cardionephritic." The first is usually the more accurate characterization.

5. *Patients with congenital abnormalities of the heart.* These usually give an electrocardiogram with right axis deviation of Q R S and an unusually large voltage of the Q R S group. Most of the congenital abnormalities throw a greatly increased strain upon the right ventricle, the resulting hypertrophy affecting the size and direction of the waves of the group. However, neither patent foramen ovale nor intraventricular band change the electrocardiogram. Hearts with congenital abnormalities often have variations in the distribution of the auriculoventricular conduction system. These variations occasionally cause abnormal duration and notching of Q R S, as is seen in Figure 23.

Congenital transposition of the heart, its apex being turned toward the right instead of the left, gives rise to an electrocardiogram suggesting right axis deviation by the small or absent R and deep S in Lead 1, but can be distinguished from this by the fact that not only is the predominant wave of the Q R S group directed downward in Lead 1, but the P and T waves also are downward in that lead. It is as if a normal Lead 1 were turned upside down; and this is practically what it is, for the right-arm wire, which is normally nearer to the auricles and the basal part of the ventricles is now, because of the transposition of the heart, nearer to the apical part of the ventricles. Likewise the left-arm wire, instead of being nearer to the apex, is now nearer to the base of the heart. The currents in the heart develop normally, but, owing to this different leading off from the heart to the galvanometer, the curve by Lead 1 is turned upside down, that by Lead 2 is what we ordinarily obtain by Lead

3 and that by Lead 3 is the ordinary Lead 2 considering its true relation to the heart. A reference to the diagram of Figure 1 will make this plain. The typical electrocardiogram described will aid in distinguishing a congenital transposed heart from one which lies on the right side, because of pleural or mediastinal disease. The latter conditions will not cause the typical abnormal features in the record.

6. *Patients with exophthalmic goiter.* These are very likely to have abnormalities of the electrocardiogram, but there are many whose records are quite normal. The more definitely toxic cases are likely to give abnormal records. The most common abnormality is an increased height of the T wave. Some develop paroxysmal tachycardia, and more than a few, particularly cases of long standing, develop auricular fibrillation. The chronic fibrotic degeneration which makes its appearance in long-standing cases often leads to one or more of those significant abnormalities of the ventricular waves described in Chapter IV.

In summary of this discussion of the occurrence of abnormal ventricular waves, it is to be noted that they are especially frequent in the chronic fibrotic heart. This was to have been expected from the experience with autopsies of cardiac cases, for the chronic fibrotic hearts are those which show the most marked changes in the muscle. We should never make a diagnosis of normal myocardium without having obtained a normal electrocardiogram, and we should be very cautious about diagnosing myocarditis if normal ventricular waves are found. Under such circumstances all other causes of cardio-respiratory embarrassment should be carefully excluded before diagnosing myocarditis.

Abnormal ventricular waves give an indication of disease of the ventricular muscle much as abnormal heart sounds indicate disease of the heart valves. Like the heart sounds, too, the electrocardiogram must be considered as an integral part of the whole clinical picture. The importance of the record is great, but nevertheless, taken by itself it can never form a sufficient basis for the diagnosis or prognosis.

CHAPTER IX

THEORY OF THE ELECTROCARDIOGRAM

Certain fundamental theses remain common to all theories of the electrocardiogram, and a preliminary enumeration of these will be profitable.

1. The reason that the cardiac contraction causes a deflection of the galvanometer is that the contracting muscle fibers acquire an electrical potential different from that of the noncontracting fibers. If the potential is increased in one area a deflection will result, while if it is diminished in the same area there will be a deflection in the opposite direction. If it is everywhere equally increased there will be no deflection, because a deflection results only from a difference in potential. If the potential is increased unequally in different parts a deflection will result, for here again there is a difference in potential, and this causes a flow of current through the lead wires and the galvanometer string.

2. The electrical record at any instant of time is the sum of all the electrical effects present at that instant within the heart; i.e., an upward wave would be due either to a single potential in such a direction as to cause an upward movement, or to the simultaneous presence of two or more potentials in opposite directions, those tending to cause an upward deflection being the larger or more numerous. In like manner the absence of a deflection would be due either to the absence of any potential within the heart, or to the presence of two or more at once, but in opposite directions and of exactly equal value. As an example of this, the quick turning downward of the R wave which produces its sharp peak might be due either to the cessation of the potentials which produced the rise of R, or to their neutralization by potentials with an opposing direction.

3. The two occasions during the electrocardiographic record when there is no deflection are considered to be due to quite different causes. The approximately level instant after S, and before the rise of T begins, is due to a balance of potential, because every part of the ventricles is contracting; while that after the T wave is completed is due to the absence of any electrical effect from the heart because there is no contraction.

4. The electrocardiogram obtained by the three standard leads best represents those changes within the heart which occur in a vertical plane transverse to the body and passing through the shoulders and left hip. This is the plane represented in Figure 1. If potentials occur in another plane, they will be represented in the plane of the standard leads by a value equal to their projection upon this plane.

5. The record obtained by the three leads does not represent the full value of the electrical potentials within the heart, because of the many opportunities of short-circuiting afforded by the body tissues between the heart and the limbs. Each wave is correct in proportion to the others, however, for the short-circuiting opportunities are the same for all.

6. The chambers of the heart act as two separate electrical units, the auricles together and the ventricles together. The curve of the auricles is independent of what is going on in the ventricles, and vice versa. Thus the P wave would have the same form, whether it occurred at the same time as the ventricular curve or at another time. The curve of the ventricles is in the same way independent of the action of the auricles. Two upward waves will be added and an upward and a downward one will be subtracted, but each retains its own form throughout, though it will be distorted by the superposition.

7. The electrical effects of the contractions of the auricles and ventricles are the same from beat to beat in the same heart, because the contraction always starts in the same place in each chamber and spreads along the same paths. The muscle contracts in the same manner in one beat as in

another, and the potential differences are developed in the same places and in the same sequences with each beat.

8. If the electrical curve of the auricles or the ventricles varies from time to time, it is due to the fact that the auricles or the ventricles, as the case may be, did not enter into contraction in the same way in the beats with the variant electrical curves. Either the path of the contraction wave was different, or the muscle itself was changed.

Differences in a Wave in the Three Leads

It has been pointed out that the electrocardiogram is different when obtained by different leads. This fact was at first a great stumbling-block in the advance of our knowledge of the subject. A wave which was upright in one lead was found much smaller in another, or even turned downward, and it was not until Einthoven's mathematical explanation appeared in 1908 that this enigma was clearly understood. Figure 5 contains the electrocardiograms of eight normal men as obtained by the three standard leads; and it is evident at a glance that there may be as much variation between the three leads of one person as there is between the same leads of different persons.

The explanation of this involves a good deal of mathematics, but may be outlined by a series of diagrams which should not be difficult to follow. Let us first examine Figure 44, which is like a record taken of the three leads at once, by means of three galvanometers recording upon the same photographic plate and within the same network of time lines. The speed of the photographic plate is much magnified as is also the distance between the horizontal lines, so as to facilitate careful measurement. A record of the same electrical potential within the heart thus falls upon the same vertical time line in each of the three leads. On measuring the height upon the same time line in each lead, it will be found that the three leads never have the same excursion at the same instant, though any two leads may be alike. The electrocardiogram may be considered as composed of a succession of brief electrical effects such as the ones we have measured.

Why is it that the same identical electrical potential within the heart is recorded differently in different leads?

Einthoven demonstrated that the three leads may be considered to form an equilateral triangle standing on one

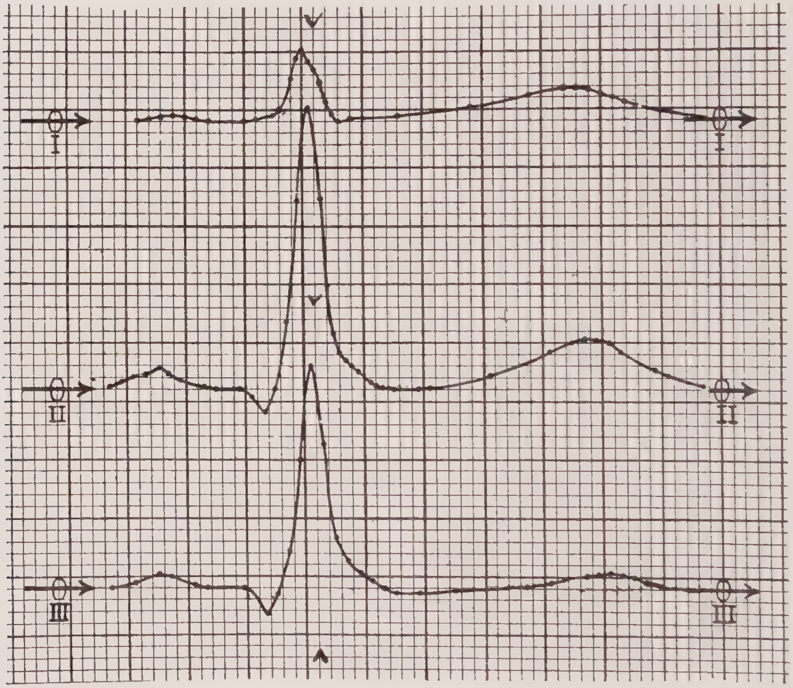

Fig. 44. From Fahr to show the electrocardiogram of the three leads in the same system of time lines, as if taken synchronously. (*Heart*, 1912, iv, Fig. 11, p. 162.)

The space between horizontal lines represents, as usual, .1 millivolt, but the space between vertical lines represents .01 second. The record is represented as if taken four times as fast as usual, hence the curves are spread out.

of its angles, and having the heart at its approximate center as shown in Figure 1. The leads do not form an exact equilateral triangle but the error from considering that they do so is a negligible one. Let the arrow through the heart in Figure 1 represent a potential within the heart, its direction representing the direction of the potential and its size the size of the potential. When such a potential is recorded by a galvanometer from leads placed about it forming a triangle, as do the three standard leads, each lead will record a proportional amount of the electricity. The proportion of each lead can be determined by projecting the arrow representing the potential perpendicularly upon that side of the triangle which represents the lead. This projection is shown by the dotted perpendiculars drawn from each end of the arrow to the sides of the triangle, and it can be seen that the length of the projection is different in each lead. Note also that if the potential within the heart is perpendicular to one of the leads, its projection upon that lead will be zero, and upon the other two leads, equal.

Since the three leads form a triangle about the heart, an electrical potential within the heart could not possibly be represented in the same way upon all of them. It is as if we should look at an approaching railroad train from three different directions, each direction being represented by the position of one of the sides of the triangle. If we look straight at the end of the train, then it will not appear to have any direction, i.e., to left or right, nor any length. From any other two directions, however, its length and the direction of its motion will be evident. If, instead of being straight in front or behind the train we are even a little to one side, then it will appear to move toward the right or the left, as the case may be, and its length will appear greater and greater the more it deviates from the end on direction. Only when it is running at right angles to our line of vision can we appreciate its true length. This is the condition which corresponds to the electrical force being parallel to the line of a lead.

As has been mentioned already, the amount actually recorded in the leads will be less than it should be on account of the short-circuiting within the body, but the proportional representation in the three leads will be correctly maintained. To represent the effect of short-circuiting, the dotted lines from each end of the arrow to the sides of the triangle of the figure should be converging instead of parallel as they approach the sides.[1]

The flow of this current from the heart through the limbs to the extremities is indicated in Figure 1 by the line of arrowheads for Leads 1 and 2, and the direction of flow through the galvanometer is represented outside the triangle

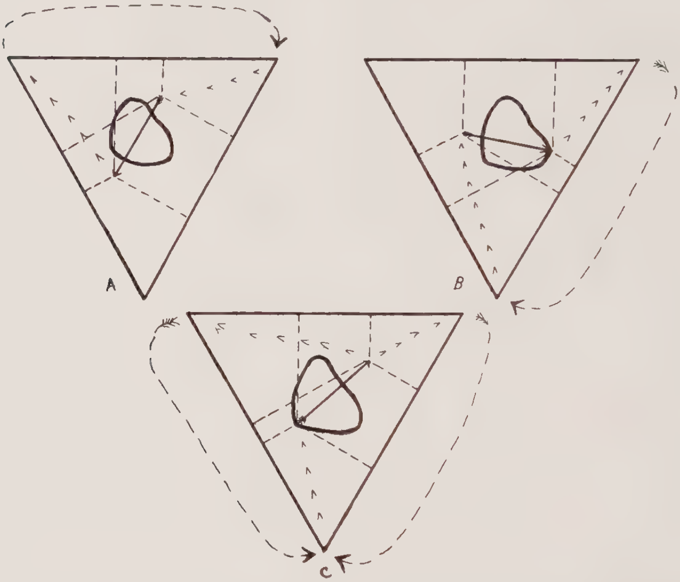

FIG. 45. Representing a direction within the heart of a current that will produce deflections in the three leads of three different sorts: A = direction to produce *downward* deflections *in Lead 1* and *upward in Leads 2 and 3*; B = direction to produce *downward* deflections in *Lead 3* and *upward in Leads 1 and 2*; C = direction to produce *upward* deflections *in Lead 1* but *downward in Leads 2 and 3*.
The triangle, heart and arrows are in the same manner as in Figure 1.

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[1] We obtain, from an experiment by Fahr, an idea of the relation of the size of the potential within the heart to the potential recorded in the leads. He placed two electrodes in the heart of a cadaver and caused a potential difference of .2 volt between them, meanwhile taking a record of the resulting deflections by the three leads in the usual way. His records showed the following values:

Lead 1 = 1.0 millivolt
Lead 2 = 4.6 millivolt
Lead 3 = 3.6 millivolt

a proportion of 200:1, 200:4.6 and 200:3.6 for the three leads, which would indicate that in the largest lead we record about 2.3 per cent of the heart's potential. Fahr and Weber. *Deutsch. Arch. f. Klin. Med.*, 1915, cxvii, 361.

by the dotted arrows for all leads. The feathered end of the arrow is toward the limb by which the current leaves the body and the head toward the limb by which it reenters. A current like that represented would cause an upward deflection of the record in each of the three leads, because of the standard method of connecting the wires from the limbs to the galvanometer.[1] This is the direction of current for the largest excursion of the Q R S group when its electrical axis is normal.

Figure 45 A shows a current within the heart whose direction is rotated *toward the patient's* right as compared with the current of Figure 1. This would cause a downward deflection in Lead 1 and an upward deflection in Leads 2 and 3. The changed direction in Lead 1 is because the direction of the potential within the heart causes the current to flow through the galvanometer from right arm to left arm instead of the reverse. Figure 45 B shows a current within the heart which is rotated toward the patient's left as compared with the current of Figure 1. The deflections will be upward in Leads 1 and 2 but downward in Lead 3. The Lead 3 deflection is downward because the flow through the galvanometer is from arm to leg instead of from leg to arm as in Figure 1. Figure 45 C shows a current within the heart which causes a downward deflection in both Leads 2 and 3, while that in Lead 1 is upward. This current is rotated still farther toward the patient's left.

EINTHOVEN'S LAW OF THE VALUES IN THE THREE LEADS

Einthoven showed that there is a mathematical relation between the size and the direction of the movements in the three leads resulting from any potential within the heart. If the deflections in the leads are measured at the same time instant it will be found that:

$$\text{Value Lead } 1 + \text{value Lead } 3 = \text{value Lead } 2.$$

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[1] The wires from the limbs are connected to the galvanometer so that a current passing through it from left arm to right arm or from the leg to either arm will cause an upward deflection in the record (Chap. I, p. 3).

This is true for positive values (upward deflections) negative values (downward deflections) and for combinations of positive and negative values.[1] If this formula is applied to measurements made upon the same time lines in Figure 44 it will hold good with a very slight error. So then, if we can decide upon *the parts of any record which are simultaneous in the three leads* we shall find that they *fulfil this formula.*

If we know the size and direction of the deflection in the three leads we can reverse the process of Figure 1 and construct the size and direction of a *hypothetical electrical force* within the triangle which would produce deflections in the leads like those we have measured. This hypothetical force was called by Einthoven the *manifest potential* (Em)-- as opposed to the *evident potential* (e) which is that recorded in the leads. The size of the manifest potential does not make allowances for the loss due to short-circuiting within the body. It is a hypothetical value and not a potential which truly exists anywhere; but still we can use it and compare its size and direction on different occasions, because it bears a certain relation to that true potential within the heart which we are unable to measure (footnote on p. 168). The ratio between the true potential and the manifest potential may not be a constant one. It is possible, even likely, that there may be different degrees of short-circuiting within the body in different individuals, and even in the same individual this may be varied by certain variable conditions outside of the heart, such as emphysema or pericardial fluid. There is at present no definite knowledge on these points.

The Angle Alpha. In designating the direction of potentials within the heart, Einthoven considered all directions in relation to the horizontal which is parallel to Lead 1. The horizontal toward the patient's left he called 0°; the 180° above this were given negative (−) values; the 180° below were considered positive (+) values. Thus 180° is horizontally to the patient's right; +30° is downward to the left and −150° is upward toward the right, exactly opposite to +30°.

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[1] See footnote, page 168, for the figures obtained with an artificial potential within the heart.

Figure 10 represents this idea diagrammatically. The circle is divided into segments of 60° each, and within each segment the sign opposite each lead indicates the movement which would be caused in that lead by an electrical force within the heart with a direction parallel to any of the radii lying within this segment.[1] If the force has a direction parallel to the dividing line between two segments, it will be perpendicular to the direction of the lead and the deflection will be zero in the lead to which the force is perpendicular. If the direction is nearly parallel to a dividing line (perpendicular to a lead) then there will be but a small value in the lead, an upward (+) deflection if it is on one side and a downward (−) deflection if on the other side of the perpendicular.

The analogy to the train is to be recalled, for the potential causes no deflection in the lead toward which it is exactly directed; just as the train has no length when viewed end on. We see that *variations in the direction* of the potential within the heart will cause changes in the *relative size* of the excursions in the three leads, and *in their direction,* whether upward or downward. *Variations in the size* of the potential within the heart will cause changes in *the size* of the waves of all the leads proportionately, but *no change in the direction* in any lead.

THE ELECTROCARDIOGRAM A SERIES OF VECTORS

The electrocardiogram is the record of an electrical potential which is constantly varying in size and direction. It may be considered as the record of a *succession of different potentials,* each one differing slightly from the preceding one, just as a motion picture is a record of a moving object, each separate view being slightly different from the others. A force having both size and direction is called a *vector,* and it may be said that the electrocardiogram is composed of a series of *electrical vectors*, each one occupying but the briefest instant. We can determine these vectors from the record by the mathematical process explained in detail in the appendix. For example, on the time line designated by the arrow in Figure 44, e 1 = 4.5 mm., e 2 = 21 mm., e 3 = 16.5 mm. Using the table in the appendix we find that these deflections must have been due to a potential at + 78° with a manifest value of 2.2. millivolts. The vector for the next time line (e 1 = 1.7 mm., e 2 = 9 mm., e 3 = 7.3 mm.) is .95 millivolts at + 79°, and so on throughout the whole of the electrocardiogram.

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[1] It must be clearly understood that the electrocardiogram does not record actual potentials which exist in any part of the heart, but that the record expresses the net result of the many potentials which exist simultaneously in the different parts of the heart. Many of these potentials are undoubtedly in opposing directions, so that their values tend to neutralize each other.

During the P wave of Figure 44 the potential first increases and then decreases in height, and the angle of the vectors gradually changes from about + 90° to about + 30°, so that the vectors change in a counter-clockwise direction toward the patient's left. During the Q R S group the potential first increases and then decreases in size, the direction of the vectors meanwhile varying irregularly in a clockwise direction. During the T wave the potential rises and falls, but there is no tendency toward a regular variation of the direction of the current.

This increase and decrease of the *size* of the vectors of P, Q R S and T is found in all electrocardiograms. It is usually a regularly progressive change but at times is found to be irregular. The variation in the *direction* of successive vectors differs also in different records. This is particularly the case with the vectors of the Q R S group. Sometimes the rotation during Q R S will be regularly in one direction, sometimes regularly in the other, and sometimes it will be quite irregular, the angle pointing now one way and now another without any predominant trend.

Records like those of Figure 6 usually show a regular rotation of the vectors of the Q R S group in a clockwise direction, starting at about − 40° and ending at + 120°. Records like those of Figure 7 usually show a regular rotation of the vectors of Q R S in a counter-clockwise direction, perhaps starting at about + 90° and ending at about − 20° or − 60°.

Records A and B of Figure 5 would probably show a rotation with the clock, and Record G against the clock, but it is rare for normal records to show such a smooth uninterrupted sweep of the vectors as do the records of right or left axis deviation of Q R S.

Mann has devised a mathematical method for constructing a graph which he calls the *monocardiogram*. This graph takes its form according to the successive variations in the size and direction of the vectors of the electrocardiogram. It introduces nothing which is not already present in the record obtained by the three leads, but it makes for a more ready appreciation of the variations in the size and direction of successive vectors. It seems likely that along this or a similar line much may be learned of the normal and abnormal electrocardiogram which could not be obtained by a simple inspection of the record by the three leads.

Lewis has shown in the dog that when the branch of the auriculoventricular bundle passing to one ventricle is cut across, the electrocardiogram for the first .04 or .05 sec. will represent only the activity of the ventricle whose bundle branch is intact. After this interval the other ventricle also becomes active, receiving its stimulus through the muscle of the interventricular septum from the first one. He showed that during the time of sole activity of the right ventricle the vectors steadily increase in size and rotate uniformly in a clockwise direction as in Figure 46 D. Lewis called the record of this interval the dextrocardiogram. The activity of left ventricle alone he called the levocardiogram, and showed it to consist of vectors which steadily increase in size and rotate uniformly in a counter-clockwise direction as in Figure 46 L. He found that by combining mathematically the values of the vectors of the dextro- and levocardiogram from the same animal he was able to reproduce a Q R S group practically identical with the normal curve of the dog in question.

Human dextrocardiograms and levocardiograms, as obtained from records indicating a lesion of one bundle branch, also show a progressive rotation of their vectors in a

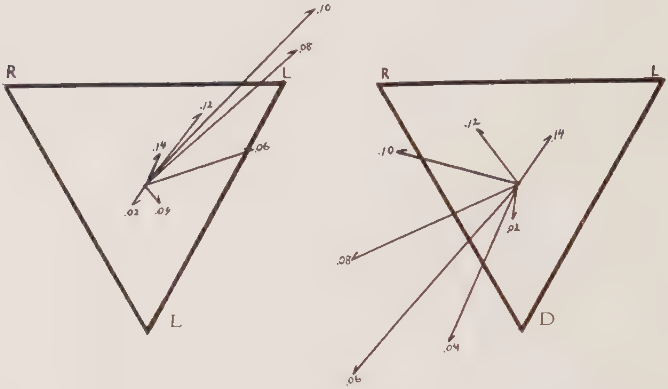

FIG. 46. Direction and size of the vectors of the levocardiogram and dextrocardiogram of Figure 47.

The triangles represent the leads, as in Figure 1. The direction of the arrows represents the angle which the vectors make with the horizontal. The length of the arrows represents the size of the vector (Em). The figures indicate the time in seconds from the beginning of the Q R S group. These diagrams are constructed from the figures in the table (p. 175).

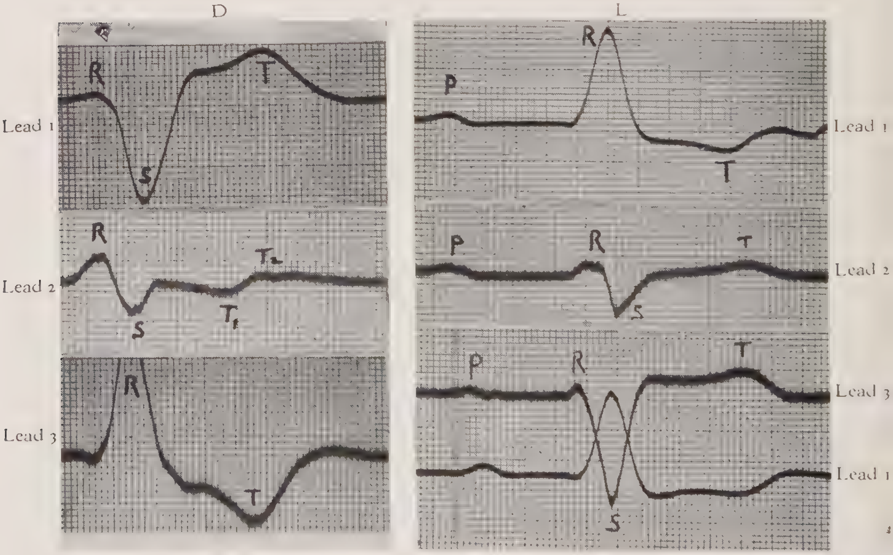

FIG. 47. The three leads are mounted as if taken synchronously. The speed of the photographic plate was such that each of the time lines represents .01 sec.

D. A premature beat arising in the right ventricle—dextrocardiogram.

L. One of the usual ventricular complexes of this patient, indicating right bundle-branch block—levocardiogram—with slightly prolonged P-R interval (.23 sec.). The record of Lead 3 has superimposed a record by Lead 1, which can be seen to be like Lead 1 of this illustration.

clockwise or counter-clockwise direction, just as do those of the dog.

Figure 47 L is a human electrocardiogram indicating a right bundle-branch lesion (levocardiogram) and 47 D is a record of a premature beat starting in the right ventricle of the same patient (dextrocardiogram).

The table below gives the angle and the value of the vectors at successive intervals of .02 sec. throughout the complexes of these two curves. Synchronous points of time in the three leads were determined by taking Lead 1 and Lead 2 simultaneously upon the same photographic plate, by means of two galvanometers, and then Lead 1 and Lead 3 simultaneously.[1]

Figure 46 was constructed from the figures in this table. Figure 46 L, the levocardiogram, shows a regular counter-clockwise rotation of the vectors, while 46 D, the dextrocardiogram, shows regular clockwise rotation.

TABLE I

Time from beginning of Q R S	47 L		47 D	
	Angle of vector	Size of vector (Em)	Angle of vector	Size of vector (Em)
.02	120°	2.0	100°	3.0
.04	52°	2.0	114°	15.0
.06	− 18°	10.0	132°?	22.0?
.08	− 42°	18.0	156°	16.0
.10	− 46°	22.0	− 164°	11.0
.12	− 52°	8.0	− 127°	6.0
.14	− 66°	3.0	− 56°	5.0

THE ELECTRICAL BASIS OF THE PRODUCTION OF NOTCHING

If the vectors of an electrocardiogram rotate irregularly by jerks while approaching the direction perpendicular to a lead, and their height increases regularly at the same time, the resulting wave will show a more or less pronounced notching, or a thickening or slurring of the ascending or descending line. Figure 48 shows diagrammatically how an irregular rate of rotation of the successive vectors can cause notching or slurring of the descending limb of R in Lead 3, which is the lead of small excursion for the Q R S group represented. Notching or slurring caused in this way is always found either in a lead of small relative excursion, or near the base line of one of large relative excursion. If the vectors rotate irregularly while leaving the direction perpendicular to a lead, their height decreasing the while, then the situation is the same as that in Figure 48, except that the timing is reversed.

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[1] This was done at the physiology laboratory of the College of Physicians and Surgeons, Columbia University, under the direction of Dr. H. B. Williams.

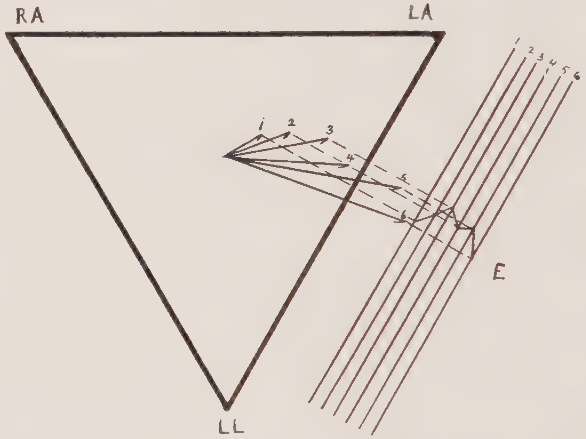

FIG. 48. Showing how notches may arise in one lead when the successive vectors rotate toward the perpendicular faster than they increase in size; or vice versa, away from the perpendicular faster than they decrease in size. In this case the notch would be in Lead 3.

The numbered radii are the vectors at successive time instants. The curve is replotted from the vectors upon the series of lines at the side of the triangle, each of these representing successive time instants corresponding to those of the numbered vectors. This replotted curve, E, would be the electrocardiogram if the electricity in the heart were as represented by these vectors.

A change in the direction of rotation of successive vectors, whether from clockwise to anti-clockwise or vice versa, may cause notching or slurring near the peak in the lead of small excursion and perhaps also near the base line in a large lead. This situation is diagrammed in Figure 49. The small vibratory Q R S group which is so common in

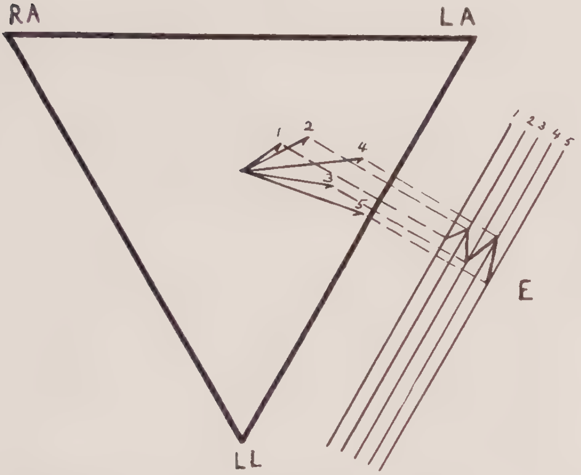

Fig. 49. Showing how notches may arise in one lead when the successive vectors do not rotate regularly, even though they increase or decrease regularly in size. Here also the notching falls in Lead 3 with the vectors used.

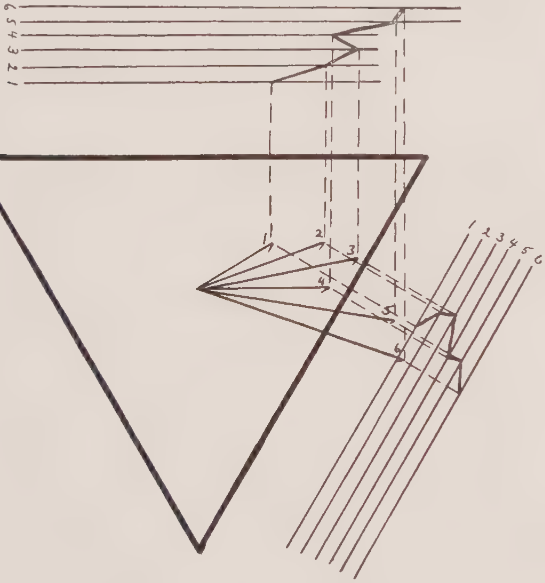

Fig. 50. Showing how notches may arise when the successive vectors do not vary regularly in size, even though the rotation is regularly in one or another direction. Here the notching is in Leads 1 and 3, but most in Lead 1, which is more nearly parallel to the vectors.

Lead 3 is usually caused by this sort of irregular rotation of the vectors.

Notching or slurring may appear, even though the rotation of the vectors is regular and constant, if the size of successive vectors varies irregularly. Figure 50 shows this condition. Notching or slurring from this cause will be more evident in the lead of large relative excursion, which is the one more nearly parallel to the direction of the vectors. This is in contrast to the notching or slurring produced by irregular rotation of the vectors, which shows most plainly in leads of small relative excursion or near the base line of larger leads.

These mechanisms of notching apply either to P or to the Q R S group. Such variations in the development of the potential depend upon the manner in which the contraction spreads over the muscle. The notching of Q R S, which appears in a small lead or near the base line of a large lead, is due to irregularities in the rotation of the vectors, or to lack of coincidence in the rates of rotation and of increase in size. This cannot be considered to indicate an abnormality of the muscle. The notching which appears in the Q R S group in large leads, usually near the peak of the wave and usually appearing in two or three leads, is not found in records from normal hearts. It is due to irregular variations in the size of the successive vectors and must be considered to indicate an abnormality of the spreading of the contraction in the muscle of the ventricles. Notching near the peak of the P wave, however, is commonly found in normal records in two or more leads, and is due to irregular variations in the development of the auricular current.

When the variation in the size of the successive vectors is irregular, coincident irregularity of rotation of the angle may increase the notching, or may partly counterbalance it. The two mechanisms may work with each other or against each other.

Notching may result from the sudden addition of the potentials of a relatively large area of muscle newly affected by the contraction wave, and it seems likely that this is the origin of the notches at the peak of the curves obtained after a lesion of one bundle branch (Figs. 12 B and 13 B). The potential from the first ventricle to contract is suddenly modified by the potential developed when the contraction spreads to the fibers of the other ventricle. Large lesions of the Purkinje system or large areas of myocardial degeneration may also produce notching of Q R S, the electrical effect of a large area of muscle failing to occur at its proper time. Notching at or near the peaks of the Q R S waves in their large leads is practically always due to an abnormality affecting Purkinje tissue or ventricular muscle. Notching near the base line in large leads or near the peak in small leads is usually not due to muscle abnormality.

It is true that these statements about the rotation of the vectors and notching are founded upon a relatively small number of records which have been examined in this way, but they are theoretically well founded and records have not been observed as yet which contradict them.

Clinical Application

For clinical purposes it is unnecessary to work out the basic potential changes in this way. These changes all lead to variations in the direction or form of the waves of the electrocardiogram in the three leads. The investigator who wishes to determine the significance of newly recognized abnormalities in the form of the electrocardiogram must have these facts clearly in mind; but the analysis having once been done need not be repeated each time the same abnormal form of the record is met with. Like variations in the heart's current will always produce like abnormalities in the three leads, so that the clinician need only be able to recognize the abnormalities appearing in the form of the curves obtained by the three leads.

Chapter X

DESCRIPTION AND OPERATION OF ELECTROCARDIOGRAPH

The essential units of the electrocardiographic outfit are:

1. A source of light.
2. The galvanometer itself.
3. A camera designed to record photographically the movements of the shadow of the galvanometer string.
4. A device for recording units of time along with the string movements, so that the speed of the waves may be measured.
5. A system of resistance coils and switches to facilitate connecting the patient with the instrument and standardizing the galvanometer.

The physical principle upon which the Einthoven string galvanometer is based is the same as that used in the electric motor. In each instrument movement is produced by the action of one magnetic field upon another. There is a very powerful electromagnet produced by coils of wire carrying a current from storage batteries, or some other steady source of current. The coils are placed about an iron core which ends in large iron "pole shoes." These point towards each other and are tapered so as to concentrate and intensify the magnetic field.

The moving part of the instrument is a very fine filament called the "string," which carries the current to be tested. It is made of platinum or better, of quartz, with a thin plating of silver or gold on its surface. The string is about .002 mm. in diameter and may have an electrical resistance of from 1000 to 15,000 ohms.

The string is held in the center of the magnetic field between the narrowed edges of the pole shoes of the magnet. It runs in a direction parallel to these edges, as can be seen in Figure 51 where *S-S* represents the string, and the pole shoes are marked *N* and *S* respectively to denote their magnetic polarity. A current passing from above downward through the string will induce a magnetic field about it with the direction shown by the dotted circle about the upper part of the string, while a current passing from below upward will induce a magnetic field about the string in the opposite direction. In the case of a current from above downward the interference of its induced magnetic field with that between the pole shoes will be on the side toward the person who is looking at the illustration, for the flow of the field between the pole shoes is from *N* to *S*. The movement of the string in this case will be away from the observer, out of the plane of the page. If the current passes in the other direction through the string, the movement will be toward the observer, because the magnetic field induced about the string is reversed, and the interference is on the opposite side.

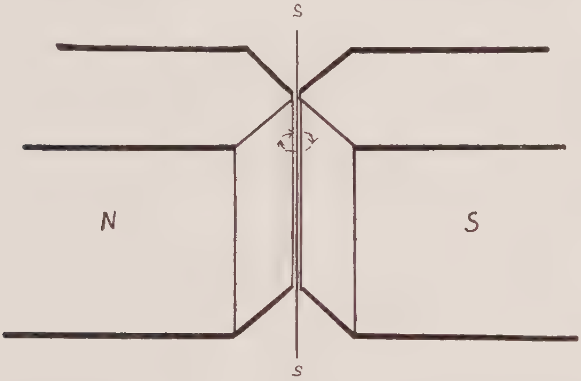

FIG. 51. The galvanometer showing how the string lies between the pointed ends of the pole-shoes of the magnet. *S-S* is the string, *N* and *S* the two pole-shoes of the magnet, *N* the + and *S* the − pole. If a current passed through the string from above downward, the induced magnetic field about the string would have the direction of the dotted arrow. This would move the string backward away from the observer.

The movements of this filament will be greater as the current is stronger, and as the tension of the string is slackened by bringing its two ends closer together. A slack-

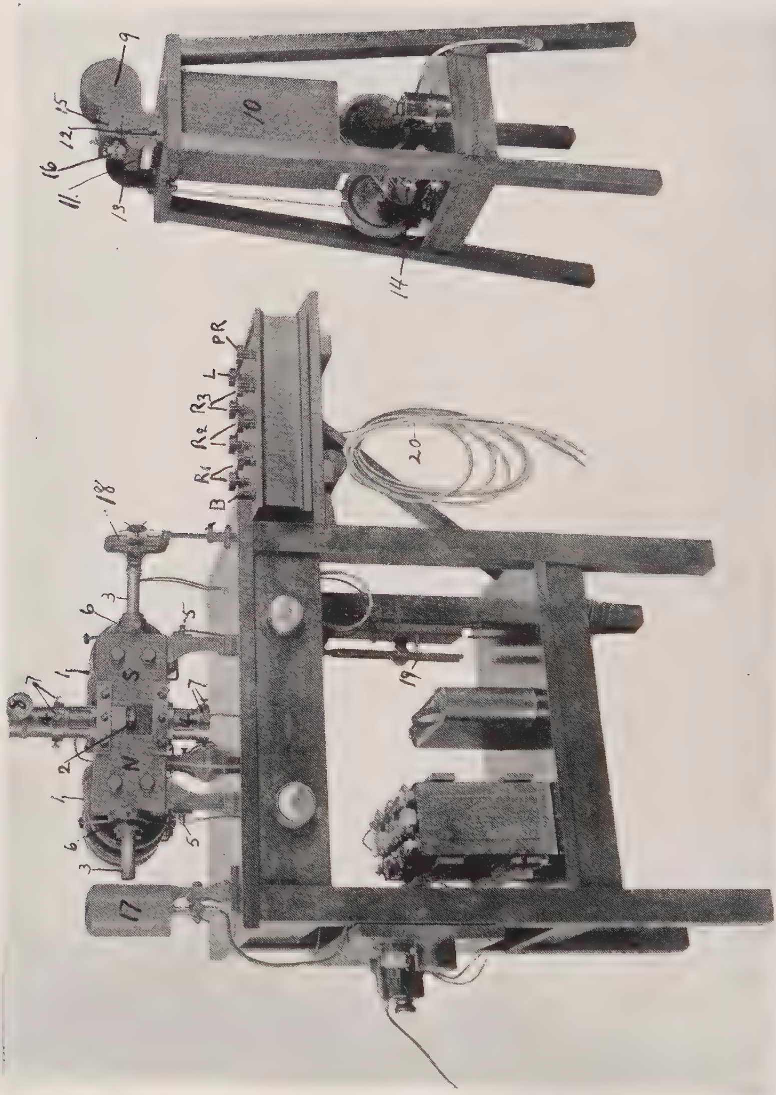

Fig. 52. Large model of the Williams-Hindle electrocardiograph.

ened string will always move more than a tense one with the same current.

The movements of the string in this galvanometer are too small to be recorded without magnification, so the pole shoes are bored to allow for the insertion of microscope tubes (Fig. 52, 3). One of these contains a condenser objective to bring the light to a focus upon the string, and the other an objective for magnification of the shadow of the string, and an eyepiece for its projection upon the photographic recorder.

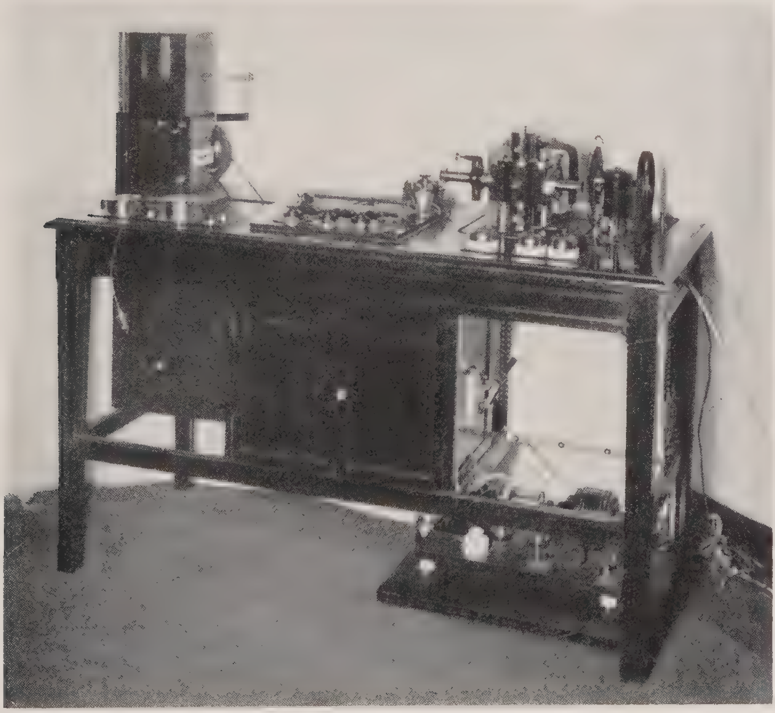

FIG. 53. The Boulitte electrocardiograph.

Many models of the string galvanometer are on the market at present. The first made in this country was designed by Professor Horatio B. Williams of Columbia University, made at first by C. F. Hindle and at present by the Cambridge Instrument Company of London and New York. Figure 52 is a model of this instrument made by Hindle about 1923. The present models are smaller and even a portable model is available which weighs about 75 lb. and

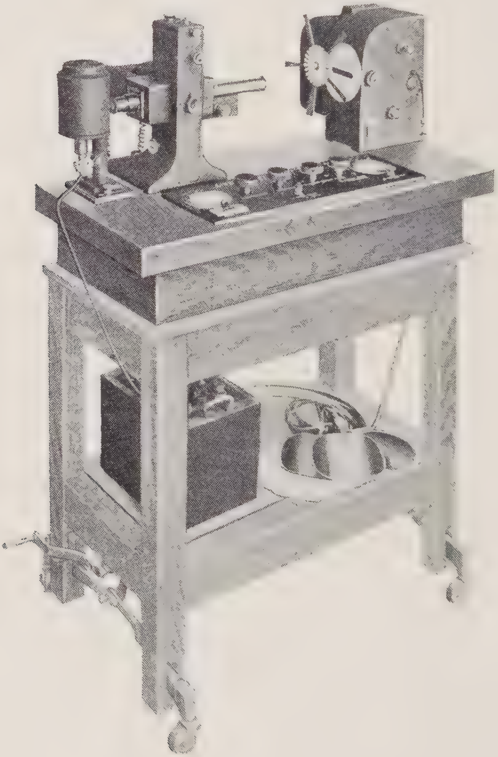

FIG. 54. The Sanborn electrocardiograph.

comes in two boxes. Satisfactory instruments are also made by G. Boulitte of Paris and New York (Fig. 53) and by the Sanborn Company of Cambridge, Mass. (Fig. 54). Portable as well as larger office models are made by both these latter companies. All of the portable models depend upon storage and dry cell batteries for the activation of the equipment so that they are entirely independent of any light circuit and may be used anywhere whether electricity is available or not.

The instrument shown in Figure 52 is a typical apparatus and can be used to explain the principles of operation which are common to all. We can see the coils of wire (1) which make the electromagnet, the large tapering pole shoes N and S held apart by brass wedges (2) placed in the angles on each side, the ends of the microscope tubes (3), the tubular brass device called the string housing (4) which is placed vertically above and below the space between the pole shoes and is used to hold the string in proper position in the magnetic field and to vary its position and tension as is necessary.

The coils of wire have a resistance of 2.85 ohms, so that the current from a storage battery which is connected to the binding post (5) will give about 3 amperes through them. With such a current passing through the coils the magnetic field is sufficiently powerful to give a correct record of the heart's electricity; but if the current should fall as low as 1.5 amperes the record will not be correct. For this reason, if a storage battery is used to activate the magnetic field, the circuit should contain an ammeter in plain sight of the operator, so that he can note promptly when the batteries are becoming weak.

In order that the microscopes may be properly aligned they are each provided with a three-screw centering device (6) similar to that used on some microscope stages.

The string housing has two objects in view. It must allow the adjustment of each end of the string so that the latter will pass through the center of the magnetic field, and must allow the string to be tightened or loosened at the will of the operator. The first of these objects is attained by the small screws (7) which, acting against springs, allow us to move the compressed fiber block to which each end of the string is attached, in any direction in a horizontal plane. This sort of adjustment is provided for both upper and lower ends of the string. To tighten and loosen the string the wheel (8) is used. Turning this acts upon the block with the upper end of the string attached, moving it upward or downward as the wheel is turning in a direction against or with the hands of a clock. Since the lower end of the string is fixed the string will be tightened when the upper end moves upward and loosened when it moves downward.

The string itself is attached at either end to a small spade-like piece of brass held in a metal piece which is insulated from the rest of the galvanometer by the above-mentioned compressed fiber block and connected to the binding posts on the back of the string housing (4). This housing is mounted upon the pole shoes in a peculiar way, because, when the current is turned on so as to activate the electromagnet, the pole shoes are drawn together slightly by the magnetic pull in spite of being braced apart by the brass wedges (2). It is necessary to prevent the string housing from changing its relation to the pole shoes during this movement, for if it did so it would carry the string with it, and move this from the center of the magnetic field. The housing is mounted on a brass plate upon the back of the pole shoes by means of a three-point suspension designed to hold it rigidly in place. It is pressed against this brass plate by a large spring.

The recording camera has a box (9) to contain a roll of film or bromide paper and another box (10) into which the film or paper passes after being exposed. Between these two is a device for making the exposure and for moving the film at a uniform rate. The light is admitted through the horizontal slit (11) and an adjustment is sometimes provided for making the slit narrower or wider. Behind the slit is a cylindrical lens which focuses the light coming through the slit upon the photographic film behind. This cylindrical lens focuses the light to a line which extends across the width of the photographic film and the shadow of the string. The shadow of the string appears as a break in this line of light. Rulings upon the lens throw their shadows on the film as it passes, thus making the horizontal lines of the records.

The film passes between two cylinders below the level of the lens which are not in contact except when the lever (12) is moved. One of these cylinders is constantly revolving at a uniform rate, being turned by the shaft (13) which is driven by a small motor (14) with a self-governing device to keep its speed uniform. This motor can be run on the house current. The speed is controlled by the operator and set at the desired point by varying a resistance in circuit with the armature of the motor. The speed remains as set with great constancy.

As the lever (12) is moved, a jerk is felt when the two cylinders make contact, gripping the film between them. We can be sure that the film is moving by glancing at the wheel (15) attached to the spindle on which the film is wound. As the lever is moved farther a click is heard, denoting that the shutter behind the slit is opened and that the film is being exposed. By moving the lever far enough to start the film and not far enough to open the shutter, unexposed film may be run into the lower box.

The pulleys on the shaft of the motor and those on the driving shaft of the camera are of different sizes, so that the successive speeds at which the film may be propelled past the camera slit are each double the next slower one, being about 12.5, 25, 50 and 100 mm. of film per sec. as the motor is usually regulated.

Above the slit (11) is a device (16) for photographing numbers on the film behind. It is an ordinary photographic shutter set for a bulb exposure. The numbers are on two celluloid discs which may be revolved at will. The numbers are exposed by pressing on the lever that opens the shutter, allowing the shadow of the numbers to fall on the end of the film behind.

Directly upon the top of the camera table is a sliding knife which must be pulled out before the lever (12) can be turned to start the movement of the film. The knife is so arranged that it pulls out a slide, opening a space in the top of the box (10) to allow the film to pass through. After the film is exposed the knife should be pushed in, thus cutting off the film and allowing it to drop into the lower box. The slide in the top of this box must be closed before the box is removed from the camera table.

The source of illumination may be a self-regulating electric arc lamp burning large carbons, or a small arc lamp with clock-work regulation which uses carbons about the size of a small pencil. An arc lamp must be used if records are to be taken at high speeds, for the crater of the carbon gives a light of great intensity. The self-regulating lamp is expensive, however, and the clock-work regulation needs occasional adjustment by hand if it is to burn steadily for four or five minutes.

A special filament bulb lamp can be used with perfect satisfaction for clinical work when the speed of the record is not to be over 25 mm. per sec. It will take readable though underexposed records at 50 mm. per sec. Though it must be replaced occasionally, the expense is negligible, for with proper handling one bulb will do for 30 records or more.

If the arc lamp is used an optic bench must be placed between it and the galvanometer to carry two condenser lenses for proper concentration of the light. There should be a water cell for cooling the light. This cell should contain weak copper sulphate solution to filter out most of the red light waves that would spoil the definition of the string shadow. The optic bench ensures correct alignment of the light, that is, it allows us by its lateral and vertical adjustments at each end to direct the beam from the glowing carbon straight into the condenser objective. This enables the operator to get a very sharp image of the string, since the beam of light can be passed exactly through the center of each lens.

With the bulb lamp we cannot use an optic bench, because the light is not sufficiently intense to be placed at the necessary distance from the galvanometer. A special lamp housing (17) is provided which allows vertical and lateral adjustment by screws and has the necessary lenses attached.

The Time Recorder. We must have a time marker of some sort recording upon the photographic film, both to serve as a check of its speed and to facilitate time measurements in the records. The simplest form is a turning fork placed upon the camera table so that the shadow of a pointer attached to one of its tines will fall vertically across the slit in front of the cylindrical lens. This produces a series of up-and-down waves of the period of the turning fork.

A Jacquet chronograph may be similarly placed with the lever vertical and will produce a line upon the record with a small peak every ⅕ sec. If the lever be placed horizontally between the source of light and the galvanometer, and its width is such that the shadow fully covers the slit of the camera, it will produce a line of shadow across the film as the lever jumps upward and another as it falls downward. Most of the illustrations in this book are records taken when using a timer of this sort. The interval from the first one of a pair of lines to the first one of the next pair is ⅕ sec. The interval between the lines of different pairs is a variable one, depending upon how high the lever goes after crossing the slit on its upward fling.

Vertical lines across the film are most satisfactory for the purpose of measuring the records, and a timer has been designed which will throw a shadow on the slit at .04 sec. intervals, every fifth line being accentuated slightly so that the space between accentuated lines is ⅕ sec. Many of the illustrations show this sort of a time marking, which, when combined with the horizontal ruling and a film speed of 1 mm. per .04 sec., gives a system of squares like those of plotting paper. This timer is shown in Figure 52 (18) and consists of a rotating wheel having four thin spokes and one slightly thicker. The shadow of the thin spokes is just sufficient to cover the width of the slit of the camera. As these pass by the slit and cover it, the light is cut off for an instant so that the full width of the film is not exposed to the light and one of the finer vertical lines is made. The thicker spoke makes the fifth accentuated line because this shadow being wider lasts for a longer time. This wheel is driven by a small motor governed by a turning fork of 50 vibrations per sec. (19). The motor makes one revolution to

10 vibrations of the turning fork and as the spoked wheel is on the shaft of the motor it is driven at a rate of 5 revolutions per sec. This timer is much the best.

The Resistance Box. When the patient's extremities are connected with the galvanometer through the resistance box there occurs a deflection of the string which is due to the difference in potential existing at the contacts of the patient's skin with the two electrodes. This is called the "skin current" and is sometimes very small, amounting to only 1 or 2 millivolts, but usually it is between 5 and 10 millivolts and it may be 20 or more. If it is large it will cause the string to move so far from the center or zero position that it will be quite off the photographic film and may be outside the circle of light thrown by the microscopes.

The objects of the resistance box are: (1) To protect the string from being forced too far and perhaps broken by this "skin current;" (2) to enable the operator to bring the string back to the center of the field of light by passing through it a sufficient current in an opposite direction to neutralize the skin current; (3) to allow the operator to produce in the circuit of the patient and galvanometer an exact potential of 1 millivolt in either direction, or of multiples of this unit, to be used in the process of standardizing the string.

The circuit within the box is shown in diagram in Figure 56 and the same lettering is used in Figures 52 and 57 which show the outside of the box as made by Hindle. *A* is a dry cell which furnishes the current. *B* is the pole shifter which enables the operator to pass a current in either direction through the rest of the circuit. *C* is a small ammeter which indicates the amount of the current. The resistance *R-1* is varied until the ammeter registers 10 and the current through *R-1* will have a value of .0001 ampere. The resistance *R-2* forms a derived circuit and the introduction of 10 ohms resistance at *R-2* will cause a difference of .001 volt across the terminals of this circuit. For each added 10 ohms there will be an added millivolt difference. If the amount of resistance used at *R-2* is enough to be an appreciable fraction of the resistance used at *R-1* the current in the primary circuit will become less than .0001 ampere, which can be noted on the ammeter. As the resistance *R-1* is usually about 14,000 to 15,000 ohms, the resistance used at *R-2* must be 300 ohms or more before there is need to consider this point. When the error arises it can be corrected for by varying the resistance at *R-1* so as to bring the reading of the ammeter again up to 10.

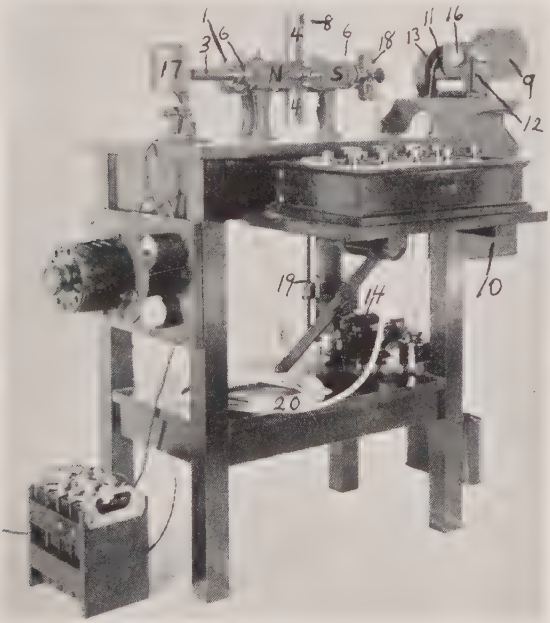

FIG. 55. Small model of the Williams-Hindle or Cambridge electrocardiograph.

At *P R* are the resistance coils which form the protecting resistance. When the needle of this switch points to *I N F* the circuit between the box and the galvanometer is open and the string is protected from all outside currents. With the needle at 2, 1,000,000 ohms are placed in circuit with the patient and galvanometer; with the needle at 1 there are 10,000 ohms and at *o* there are none. The high resistances protect the galvanometer string, as the patient is turned in, from the full impact of a large skin current or of any extraneous currents which may be in the circuit through faulty insulation or poor contacts. The current will be reduced by the large resistance and the string kept from deflecting outside of the circle of light.

The wires from the three extremities of the patient are led into the box at the binding posts marked *R A* (right arm) *L A* (left arm) and *L L* (left leg). The lead switch *L*, marked *1*, *2*, *3*, *4*, is a device for connecting these wires to the galvanometer in such a way that when the needle points to 1 Lead 1 is properly connected (*R A-L A*) when the needle points to 2, Lead 2 is connected (*R A-L L*), and when it points to 3 Lead 3 is connected (*L A-L L*).

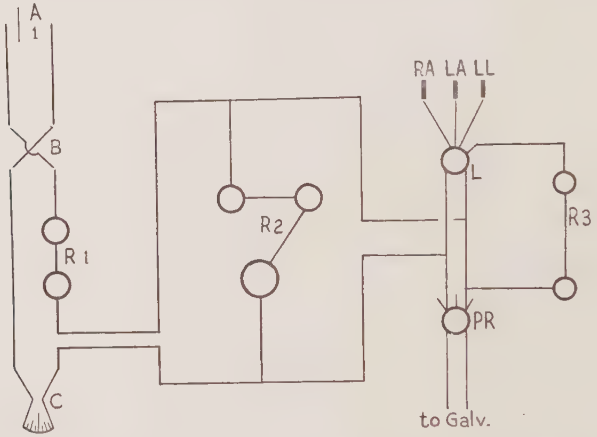

FIG. 56. Schema of the wiring of the resistance box. A, an ordinary dry cell; B, a pole changer; C, an ammeter to measure the current flowing from the dry cell through R-1; R-1, resistance on the circuit of the dry cell. Wires from the right arm (*R A*) left arm (*L A*) and left leg (*L L*) come into the lead switch (*L*) by means of which any two extremities may be connected to the galvanometer. The protecting resistance (P R) is placed in the circuit to the galvanometer. The current from the dry cell A will not flow through the galvanometer with its high resistance, but takes the short circuit afforded at *R*-2. By introducing resistance here, more and more current may be passed through the galvanometer. *R*-3 is an extra resistance which is thrown in circuit with the galvanometer when the lead switch is turned to 4 (Fig. 57).

When the needle of this switch points to 4 the patient is disconnected from the galvanometer, and the resistance *R-3* is substituted for the patient. This resistance is used for measuring by comparison the resistance of the patient and may also be used to measure the resistance of the string.[1]

The other binding posts along the back of the resistance box are quite plainly marked, the two on the left being for the + and − poles of the dry cell which furnishes the compensating and standardizing current.

In this resistance box all the coils and contacts are enclosed in a copper-lined box, all the handles are of metal and connected to the copper lining, and this lining can be earthed from the terminal marked "Earth," so that effective screening of the enclosed circuits is provided, from static charges which might be induced by the movements of the hands of the operator or other causes.

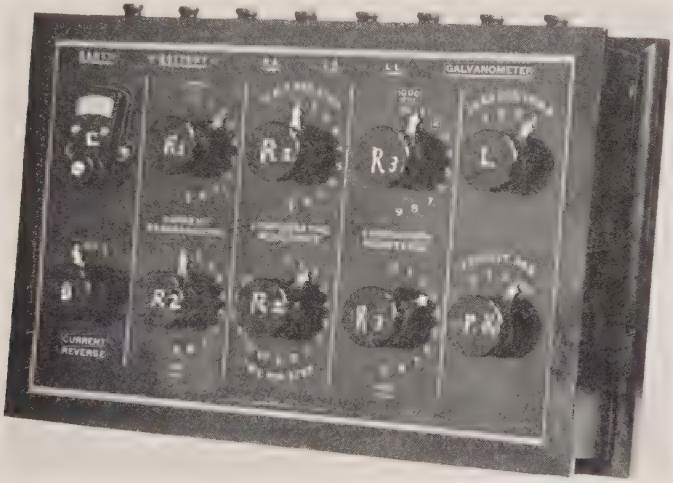

FIG. 57. The resistance box.

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[1] To measure the resistance of the string the galvanometer is turned on, the lead switch is turned to contact 4 and the protecting resistance to zero. A potential of 3 millivolts is thrown into the galvanometer circuit at *R-2*; the tension of the string is increased or decreased by turning the knob (8) of Figure 52 until the deflection of the string shadow for the 3 millivolts is exactly 3 cm. measuring on the ruled plate above the slit of the camera. Resistance is now turned in at *R-3* until the deflection of the string is reduced to exactly 1.5 cm. for 3 millivolts. The amount of the resistance necessary will be equal to the resistance of the string and can be read off from the dials of *R-3*.

INSTALLATION

When setting up the instrument it is advisable to have the galvanometer on a strongly built wooden table. It makes no difference in which direction the light passes through the microscope tubes, but for convenience it should be so that the string housing side of the galvanometer is toward the operator. The source of light should be on the same table as the galvanometer. The camera is placed one and a half meters away from the projection eyepiece,[1] and the resistance box is conveniently placed between the camera and the table holding the galvanometer, as shown in Figure 52.

Complete directions for setting up the apparatus are furnished with the instrument, so that only certain things need to be emphasized here.

All wires used about the instrument except those which activate the electromagnet should be lead covered, and it is advisable to have these coverings grounded. The grounding wire attached to the back of the resistance box should be of ample size, and the frame of the galvanometer should be grounded from the binding post provided on the rear leg. One of the ends of the string should be grounded. This may be done by connecting together the adjacent terminals on the resistance box. These groundings, particularly the last two, will prevent static charges developing upon the instrument or the string, so as to draw the string against one of the pole shoes and perhaps break it. If an alternating current is in use in the building there may be the greatest difficulty in screening the galvanometer circuit from the magnetic field about the power wires. This field is constantly varying and will induce a current in the galvanometer circuit of the same frequency as the alternations of the current, producing a continual buzzing movement of the galvanometer string. To prevent this the power wires should be enclosed in an iron pipe which will catch most of the magnetic lines of force and prevent their reaching the galvanometer circuit. The thicker the iron, the greater the protection. Similar induction trouble can arise from the wire supplying the motor of the camera or of the timer, should these wires pass near the galvanometer circuit; or it can arise from the wire to an arc light with direct current if the arc light should flicker.

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[1] Another model has a short focus projection ocular so that the camera is quite close to the galvanometer (Fig. 55).

Heavy wire should be used to connect the box to the galvanometer. If the wire is not stiff enough to prevent vibration from jarring of the table or building, its movements in the strong magnetic field of the instrument will set up currents within it, by cutting the lines of magnetic force of this field. These currents will distort the electrocardiographic record.

When inserting a string in the large Hindle galvanometer a pocket flash light will serve well to bring the string into view. It is best seen in a dark room and against a black background, and rarely can be seen throughout its entire length at once. Never attempt to insert the string until after practicing with the copper wire dummy which is provided nor without the illumination of the lamp through the condenser microscope.

Never screw in either of the microscope tubes after inserting a new string, without having the string itself plainly in view where it lies between the two objectives. Only thus can we avoid the chance of breaking it, by screwing the objective against it. It is helpful to use a hand lens to see the string as the objectives come close to it. Remember that the condenser objectives should be about .4 mm. from the string. With this arrangement the projection objective will be about .2 mm. from the string when the image is obtained.

The proper *centering of the optical system* had best be practiced with the dummy string in place to prevent possible mishaps. When the source of light and the galvanometer microscopes are in proper alignment, screwing the projection microscope (3) in and out so that the string shadow goes out of focus first one way and then the other, will result in a symmetrical widening of the shadow to either side. Likewise the edge of the circle of light will go out of focus concentrically and will continue to be circular. If the alignment of the system of lenses is improper, the string will broaden out to one side when the microscope is screwed one way, and to the other side when screwed the other way. The light circle will do likewise and will assume an elliptical form. With poor alignment it is impossible to get a sharp photographic image of the string. The alignment should be corrected by moving the source of light, the lamp or the optic bench as the case may be, to one side or the other until the string shadow and the circle of light remain symmetrical as the microscope is focused.

If lateral movement of the source of light does not correct the alignment so that focusing gives a symmetrical widening of the string shadow and of the circle of light, then the trouble must be in the centering of the microscope tubes. To align these is a nice procedure and it should not be attempted unless one is quite familiar with the structure of the instrument.

Turn off the galvanometer current and the lamp. Remove the brass wedge (Fig. 52, 2) by first loosening the screws that hold the bars across its top and bottom, then working the pins of these bars free from their holes in the pole shoes and carefully and slowly lifting the wedge, by means of the knob at its center, free from its place between the pole shoes. This exposes the two microscope objectives which can be seen projecting from the holes in the pole shoes, and if a pocket flash light is used to illuminate the string, this will be seen suspended in its place between the pole shoes. Next screw the projection microscope entirely out of the instrument, and on looking through the hole from which it came the end of the condenser objective will be seen. Use the pocket flash light to illuminate the string and center the condenser microscope by means of the three screws of the centering device (6) so that the string cuts across the vertical diameter of its lenses. Replace the projection microscope without its ocular, being careful to avoid screwing the objective against the string. Turn on the lamp and hold a card about a foot in front of the projection microscope. There will be seen on this card two circles of light—an inner bright one and an outer less bright—with the string shadow falling vertically across them. The centering adjustment (6) of the projection microscope must now be manipulated so as to make these two circles of light concentric. Replace the projection ocular and the brass wedge, and the procedure is completed.

The photographic image may fail to have sharp edges, even though it appears sharp to the observer, because of chromatic aberration in the lenses. The operator should find by experiment a point somewhere between the eyepiece and the camera at which he can focus the shadow of the string and obtain a sharp photographic image. The visual image at the distance of the camera will of course then be blurred. The visible light rays which we focus by the eye are mostly from the red end of the spectrum, and these converge from the microscope more sharply than the more actinic blue rays which produce the photographic image.

When connecting the dry cell to the resistance box it is convenient to connect its poles in such a way that turning the pole shifter to the right will enable the operator to move the string shadow to the right upon the camera by introducing resistance at *R 2*.

When connecting the resistance box to the galvanometer, the wires should be so connected, when film is used, that the normal *R* wave causes a movement in a direction toward the right-hand side of the camera slit as one faces it standing by the galvanometer. If this precaution is not taken, prints made from the negative must be printed with the film instead of the emulsion against the paper. Prints made in this way do not, of course, have as good definition as when the emulsion lies next to the paper.

When bromide paper is to be used in the camera the wires to the galvanometer should be connected so that the normal *R* wave causes a movement in the reverse direction, i.e., toward the left side of the camera slit, or the record will be upside down.

Taking the Record

The patient must first be connected with the resistance box. A flexible cable (Fig. 52, 20) containing three wires twisted together and covered with a flexible metal sheath should be used. In a hospital this cable may lead to a wall plug which connects with a cable running to various parts of the building, and records have been thus taken of patients lying in their beds. The development of portable electrocardiographs has now made this unnecessary. In the office the wires of the flexible cable are attached at one end to the contacts *R A*, *L A*, and *L L* of the resistance box and at the other by special electrodes to the right arm, left arm, and left leg of the patient.

The position of the patient is not especially important, but he should not sit slumped down in an upright chair, for this will tend to press the diaphragm upward into the thorax and to make the heart lie more transversely. The effect of this upon the electrocardiogram has been described. It makes no difference whether the upright, semi-reclining, or full reclining position is used; but subsequent records of a patient should always be taken in the same position as the original, in order to be comparable.

To connect the patient with the wires we may use one of several types of *electrodes:* a simple one consists of bandages wet with a strong solution of common salt applied about each wrist and the left ankle of the patient. It is necessary to have the salt solution nearly saturated, at least over 10 per cent. The ends of the bandages are passed over a metal plate, binding the plate to the limb. The wires are attached to the plates. A thin lead plate may be used and bent about the bandaged limb to hold it in place. Other metals would serve, but are not as convenient as the lead because of its flexibility and relative freedom from corrosion. An even more satisfactory electrode for bed patients consists of a bandage wet with hot salt solution about which is wrapped 18 inches or so of 18 gauge copper wire. This wire is connected to the lead wires. With this electrode some of the tendency to "overshooting" (page 202) is avoided.

For taking records when the patient comes to the instrument it is more convenient to use tanks containing strong salt solution, the hands and foot being placed in the liquid. Metal plates, again preferably lead, because of its freedom from corrosion, are immersed in the salt solution, and the wires attached to these. It is very important that the bandages of the plate electrodes and the salt solution of the tank electrodes be as hot as can be borne, for otherwise there may result either shivering of the patient's extremities or marked vasoconstriction of the skin vessels. The former will cause a fine vibration throughout the record, due to currents originating in the striate muscles (Fig. 5 c, Lead 1, and Fig. 12 A, Leads 1 and 2). The latter will cause the skin to have a high resistance to the passage of the current (Fig. 58 A).

Polarization does not seem to affect the usefulness of these electrodes, the probable reason being that the heart currents are so small in relation to the size of the electrode surface that the polarization which does occur at their surfaces never becomes marked enough to distort the current of the heart. The author has shown by experiment that neither the bandages with a large German-silver plate nor the tank electrodes polarize as used in electrocardiographic work; and Cohn has similarly shown that lead-plate electrodes over bandages do not polarize.

The patient is thus connected by wires and electrodes with the resistance box at the contacts *R A*, *L A* and *L L*.

To take Lead 1 the switch *L* is turned so that it points to 1, the resistance *P R* is turned from *I N F* to 2. The string shadow should be watched as this is done and it will be seen to jump to one side or the other. This movement is due to the "skin current." At times this current will be strong enough to throw the string shadow completely outside the field of light. This is often due to improper application of the electrodes, especially to getting salt solution on the contact between two different metals on one of the electrodes or to having one bandage wetter than the other. It may, however, be a feature of the vasomotor condition of the patient's limbs. The way to correct it is to reapply the electrodes, with care that the bandages are hot and equally wet.

When the operator observes the direction in which the skin current is deflecting the string, he should turn the pole changer *B* to throw the current into the box in such a direction as to oppose this movement of the string. When the dry cell has been connected as described above, the pole changer should be turned to the left if the string shadow has moved to the right, and vice versa. This will not cause any movement of the string, as the resistance at *R-2* should be at zero. The next step is to turn in sufficient resistance at *R-2* to bring the string shadow back to the center of the field of light, using first the 1-millivolt series, and if more is needed turning on one or more of the 10-millivolt units. When the shadow is approximately at center, *P R* may be turned to 1, and if the string shadow again moves far off center a sufficient number of millivolts should be turned in or out to bring it again to the center of the camera slit. *P R* is now turned to *o* and the centering process repeated if necessary. This procedure is called *compensating* for the skin current, and it will be recognized that what we have done is to turn on sufficient current, in a direction opposite to the skin current, to neutralize it completely. Remember that if over 3 units of the 10 millivolts series of *R-2* are used in compensation, the strength of the current from the dry cell will be reduced below .0001 ampere. This current should be brought up to its proper strength by removing as many 100-ohm units from the resistance *R-1* as have been introduced at *R-2*. If the electrodes are uniformly moistened and properly applied there will never be need for as much as 10 millivolts compensating current.

The operator is now ready for the next step, namely, the *standardization* of the galvanometer so that 1 cm. excursion of the string shadow will represent 1 millivolt in the circuit of patient and galvanometer. The knob of *R-2* which gives 1-millivolt units should be turned to and fro so that 1 unit is repeatedly added to and subtrateed from the compensating current. The amplitude of the resulting jumps of the string shadow is regulated by turning the wheel (8) on the top of the string housing so as to tighten the string if the jump is too great, and slacken it if the jump is too small. When the string has been adjusted so that it moves approximately 1 cm. in response to 1 millivolt, then 3 millivolts should be turned in quickly, so that any error in standardization will be multiplied by 3 and can be corrected by further adjustment of the tension of the string. With the box which has 15 1-millivolt steps the string can always be moved 6 cm. directly across the width of the slit, thus making the standardization still more exact.

If the patient is not mentally at ease there may be a continual variation in the skin current, and the string shadow will wander back and forth across the field so that standardization becomes difficult. The patient should not converse while the record is being taken, as this will usually cause the skin current to vary. We should strive for both mental and physical relaxation.

Contraction of the muscles of the extremities will cause a buzzing vibration of the string which makes it appear out of focus. A simple tension of the muscles such as might result from an uncomfortable position will be quite sufficient to cause this. The patient should be made comfortable and free from distraction of any kind.

Having *compensated* for the skin current, and *standardized* so that 1 millivolt causes 1 cm. deflection, the operator is now ready to *take the record of this lead.* The film is exposed by moving the lever (12) on the camera as far as it will go to the right. Now record the standardization by turning in and then out again 1 or 2 millivolts while the film is being exposed, and then allow three or four revolutions of the flywheel, which will give 9 or 12 in. of record after the standardization test.

Before the lead is taken it is always well to ascertain *the resistance of the patient.* This is done by turning *P R* to *I N F*, turning the lead shift knob to *4*, and then, after all the dials of *R-2* are set at zero, turning *P R* to *O*. We have now substituted the resistance *R-3* instead of the resistance of the patient; and we determine the patient's resistance by comparison with this, varying the resistance *R*-3 until 3 millivolts from *R-2* produces a deflection of exactly 3 cm. The resistance shown at *R-3* then will be the same as was that of the patient.

When measured in this way the patient's resistance should not be over 2000 ohms. If it is more than this there will result a distortion of the electrocardiogram, more marked if the bandage type of electrodes is used than with the tanks. There can be seen in the control of standardization at the beginning of each lead of Figure 58 A an *overshooting* of the deflection due to the millivolt. Compare this with the sharp angle at the end of the millivolt deflections in Figure 58 B. After the overshooting the string takes from .10 to .20 sec. to return to the level at which it finally remains. Overshooting

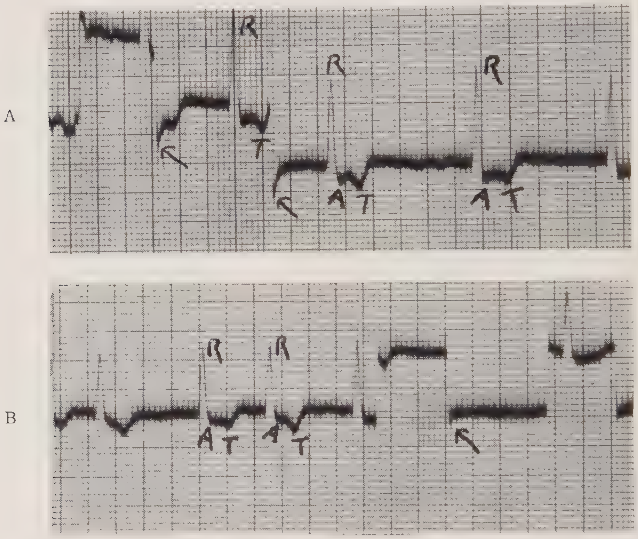

Fig. 58. Two records of Lead I from the same patient, to show the deformity of R and T due to overshooting and also that the overshooting diminishes with the resistance. The upper record was taken when the resistance was 8000 ohms, the lower, when it had fallen to 2000 ohms. The overshooting is seen in the standardization control where the arrows point. Note the greater height of R in the upper record, and compare the shape of T in the two records at the points indicated by A.

will be more marked the more the string must be slacked to obtain a permanent deflection of 1 cm. per millivolt, but the overshooting is not due to the slackness of the string. Under these conditions a potential which develops quickly as does Q R S will cause a quick deflection of more than 1 cm. per millivolt and after the deflection there will be a slow return toward the base line distorting the record of the heart's potential at that time. Thus the P, Q R S and T waves will be too large, and after each wave the record will overshoot the zero level and curve slowly back toward it. Moreover the height of a wave which begins relatively slowly, as does the T wave, will not overshoot so much and so will be relatively smaller than the quickly developing waves. When this artefact is markedly present the end of the T wave will also overshoot the zero and then return to it, or if T is downward, will curve above it, forming an exaggerated U wave.

The cause of this overshooting is not certainly known. It does not depend upon the slackness of the string, for if the two ends of the lead wires are short-circuited the application of 1 millivolt at *R-1* will cause a movement of the string without sign of overshooting, though it will be a large movement because of the low resistance of the circuit. If the knob for shifting the leads is turned to *4*, so that the resistance *R-3* is placed in circuit instead of the patient, it will be found that a large resistance must be used in this bank to bring the excursion of the string resulting from 1 millivolt to the same size as the deflection, when the patient is in the circuit. It might be 5000 ohms or more, but the application of a millivolt at *R-2* through this resistance will not cause overshooting as it does through the patient, though the tension of the string has remained unchanged and the resistance of the circuit is high.

The cause of the overshooting must lie in the patient. I believe that it is the vasomotor condition in the skin under the bandages; for it is a rare thing to find overshooting when the patient's extremity is warm, and common when it is cold. Furthermore, I have seen the overshooting disappear suddenly as the cold extremity became warm, also accompanied by a change in the skin current. Since using smaller lead-plate electrodes and the copper wire electrodes described, I have found overshooting to occur less frequently than when I used larger German-silver electrodes. It is very rare to find it when using tank electrodes. It is always found in a patient with cold extremities because of a weak failing pulse, and commonly when edema is preset.

It is my opinion that the overshooting is due to the vasoconstriction in the skin raising the electrical resistance of this structure so that it functions as a condenser surface. The large initial jump is due to the flow of current while the condenser surface of the skin is taking up its charge; the lesser amplitude of the maintained deflection is due to the flow of current through from one to the other side of the skin, because this surface is not absolutely a non-conductor. This hypothesis has not been completely investigated, but all the experiments performed as yet are in agreement with it. Especially noteworthy is the observation of less overshooting for the same resistance with the smaller electrodes, for the condenser surface under the bandage is smaller when the electrode is smaller. The skin surface in contact with the water in the usual tank electrode is also small, thus accounting for the rarity of overshooting with these.

Whatever the fundamental cause, the overshooting can usually be avoided by having the extremities comfortably warm. To this end the room must be warm or the patient covered with blankets; the bandages or the tank solution should be as hot as comfort will allow, and if still the overshooting is observed, a hot-water bottle should be placed so as to rest near the part of the extremity to which the electrode is applied. Allow from three to eight minutes after applying a hot-water bottle for a vasodilatation to take place, and the condition will usually disappear.

Leads 2 and 3 are taken by exactly the same procedure as employed for Lead 1 except that the lead shift knob is turned to 2 and 3 respectively. If it is found that the string tension does not have to be diminished from one lead to the next, we know that the resistance of the second of these leads is not more than that of the first. If the first is low, it will be unnecessary to measure the resistance of the second, for the only important feature of resistance is that it shall not be high. In time, the operator will be able to detect the overshooting due to high resistance during the process of standardization, and then the need for measuring resistance will be past.

After the third lead has been taken, and before cutting off the film, the lever (12) on the side of the camera should be moved to the first catch and left so while the flywheel makes two revolutions. This runs the end of the record from the level of the slit down to below the level of the knife, and leaves a blank unexposed piece of film in this situation behind the numbering device, upon which to photograph the number of the next record. Pressing the knife sharply in will cut off the film and allow it to drop into the box. The top of the box should now be closed and the box removed to the dark room for developing.

There are so many steps necessary in taking an electrocardiogram that it is well to establish a definite order so that nothing will be forgotten. The following will serve as a summary of the taking of a record and suggest what has been found to be a useful order of procedure:

1. Heat the salt solution or make it up freshly with hot water.
2. Apply the electrodes.
3. Turn on the lamp.
4. Turn on the current which activates the turning fork and start the time wheel.
5. Turn on the current to activate the galvanometer magnet.
6. Start the motor of the camera.
7. Photograph the number of the record upon the end of the film or paper.
8. Pull out the knife and open the top of the box to receive the record.
9. Turn the lead switch to Lead 1.

10. Compensate and standardize.
11. Measure resistance.
12. Expose Lead 1 if resistance is satisfactory.
13. Turn off protecting resistance and then turn lead switch to Lead 2.
14. Compensate and standardize.
15. Measure resistance if necessary.
16. Expose Lead 2, if resistance is satisfactory.
17. Turn off protecting resistance and then turn lead switch to Lead 3.
18. Compensate and standardize.
19. Measure resistance if necessary.
20. Expose Lead 3.
21. Turn protecting resistance to infinity.
22. Allow paper to run for two revolutions of flywheel without opening shutter.
23. Cut off paper and close top of box.
24. Turn off motor, switches of resistance box, galvanometer magnet, time wheel and light.

Difficulties in Operation

The electrocardiographic outfit needs but little attention to keep it in proper running order. Keeping the batteries properly charged and filled with water, and the time wheel and camera motor oiled, is about all that has to be done. Occasionally, however, trouble will develop, and it is well to know how to locate the most common causes so that the apparatus can be promptly put in order.

If on turning in the patient, it is found that the string is not deflected as usual by the skin current and the electrocardiogram, first make sure that the galvanometer current has been turned on, and then test another lead to see if that behaves similarly. If no deflection is obtained from any of the lead connections, turn the switch to Contact 4 and attempt to pass current through the resistance coils, as for measuring the patient's resistance. If no deflection is obtained through the resistance coils, then the string itself should be tested by touching one moistened finger-tip to each of the terminals on the string housing. The difference in potential between the two finger-tips should cause the string to jump. If it does not, then the string has ceased to conduct and must be replaced by a new one. Obviously if the string does jump when the terminals are touched in this way and has not moved during the previous procedures, the trouble must lie in the connections.

If the string shows large, very rapid, irregular vibrations while the dials of the resistance box are being turned, it is due to dirt between the contacts of the dials. This should be promptly corrected, for it may cause the string to be thrown against and stick either to the side of the slit in which it lies, or to one of the microscope objectives, so that its shadow will not appear in the circle of the light.

To clean the contacts of the resistance dials, open up the box by removing the screws on the bottom, and wipe the surfaces with a fine tissue paper, while turning the dials back and forth. Do not use emery, as the fine grit remains and makes the contact worse rather than better.

If the string suddenly disappears from the field of the microscope while the instrument is being used, it may have been due to a sudden large variation in the skin current. Turn the protecting resistance *P R* to *I N F*, and the string should reappear. If it fails to do so, its disappearance is probably due to its being stuck either to the microscope objectives or to the side of the slit. This may be due to its having acquired a static charge from defective grounding or to the above-described result of dirty contacts in the resistance box. To free the string, turn off the galvanometer current and turn the protecting resistance to zero, which will distribute any static charge still remaining. Tapping the microscopes gently with a lead pencil may now dislodge the string if it is stuck to them, when its shadow will reappear in its proper place in the circle of light. If this does not suffice, a more complicated procedure will be necessary. Turn a spoke of the time wheel so that its shadow appears in the light circle and focus the shadow sharply. Screw the front microscope forward about ¼ in. by means of the focusing adjustment and then screw it back to its former position so that the spoke of the time wheel is in focus again. If the string was stuck to the objective of this microscope it will have been dislodged by this process and will reappear in the circle of light. If not, screw the rear microscope backward about ¼ in. and then forward to its former position so that the time-wheel spoke is in focus. This will dislodge the string if it was stuck to the rear microscope. The precaution concerning the shadow of the time wheel is necessary to prevent each microscope being screwed back past its proper position and against the other one. The only time the author has ever broken a string in the Hindle instrument, it was due to omitting this precaution and crushing the string between the microscope objectives when they came together. If the string fails to reappear after the above manipulations of the microscopes, it is either stuck to the sides of the slit or is broken. Turn the string adjustment wheel for 3 or 4 revolutions so as to tighten the string. This may pull it free; but if it does not now appear in the circle of light, open the front of the galvanometer by removing the wedge, and view the string directly with the aid of a hand lens and a pocket flash light. The process of removing the wedge will often dislodge the string so that it reappears in the circle of the light. If it does not, and the two ends of the string can be seen where they extend beyond the poleshoes of the magnet at top and bottom, we know that the string is not broken. Screw the lateral adjustment (7) so as to bring the string toward the observer. This will pull it free, or will show that the string is broken.

The precautions against such sticking of the string are: (1) Keep the resistance box contacts clean and (2) never leave the galvanometer with the string unusually slackened, e.g., so that it moves more than 1 cm. per millivolt through 2000 ohms. With the small model of the Cambridge instrument the string should be left at such a tension that it will deflect 1 cm. for 1 millivolt with no resistance in circuit.

If we find that on turning in the current from Lead 1 there is no deflection of the string, while on turning in either of the other leads, there *is* a deflection, the trouble lies in the connections with the patient. If Lead 2 is right and Leads 1 and 3 are dead, then the trouble lies in the wire which enters into Leads 1 and 3 and not into Lead 2, i.e., the left-arm wire. If the trouble is inside the resistance box there will be no deflection of the string on touching moistened fingers to the binding posts of the two dead leads, the protecting resistance being turned to *0*, e.g., *R A* and *L A* (Lead 1) and *R A* and *L A* (Lead 3) if Lead 2 is functioning properly. If this procedure gives a deflection of the string then the trouble lies in the wires to the patient.

The electrocardiograph, as manufactured by the Victor X-Ray Corporation, differs from the so-called string type of electrocardiograph originated by Einthoven, both in principle and in method of operation. Instead of recording the heart's tiny current by means of a very sensitive galvanometer, the current from the heart is so greatly amplified that a less sensitive instrument will serve. In this new apparatus (Fig. 59) the recording element is a beam of light reflected from a moving mirror, instead of the shadow of a moving string as in the Einthoven model. The current from the heart passes through a three-tube resistance coupled amplifier which increases its strength approximately three thousand times. The tubes are similar to those used in radio work and besides having a long life are easily replaceable. This amplifier was especially designed to meet the needs of electrocardiographic work and amplifies the electrocardiogram without distortion. The filaments of the tubes require 5 volts for energizing and this is obtained from a storage battery which is part of the equipment. The plates of the tubes require 150 volts, and here again the necessary amount of voltage may be obtained from the dry "B" batteries.

The sensitivity control consists of two switches with five points of regulation each. One is for coarse adjustment and one for fine adjustment. They are used to cut in or out resistance across the grid of two of the tubes, which serves to regulate the amount of amplification.

The standardization switch, when pressed downward, automatically connects across the 5-volt filament circuit a resistance of such value that 1 millivolt is obtained. To obtain exactly 1 millivolt it is necessary to have the filament circuit at exactly 5 volts. This is read from the voltmeter which is across this circuit. When the storage battery is below the proper voltage, it should be recharged. In practical use the storage battery can be used as low as 4.8 volts, because the difference is so small that the effect on standardization is not appreciable. In practical use a full charged battery will operate the equipment a half hour a day for a week before charging is necessary. The dry "B" batteries

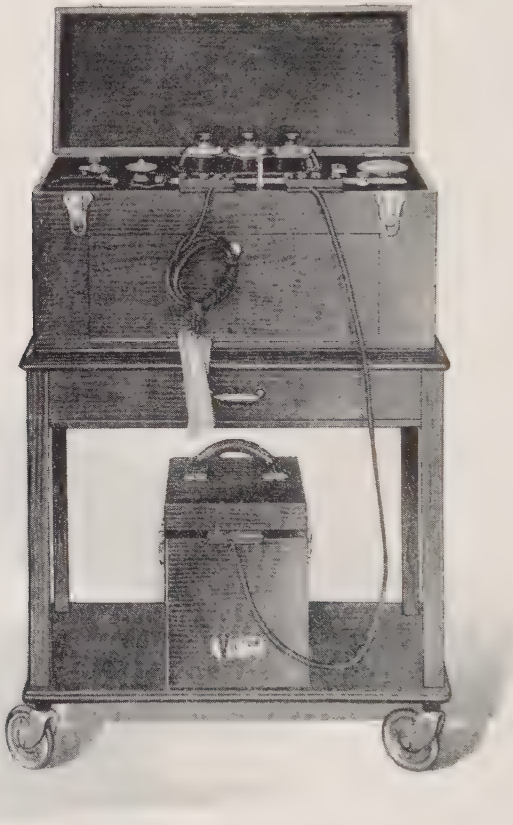

Fig. 59. The Victor electrocardiograph.

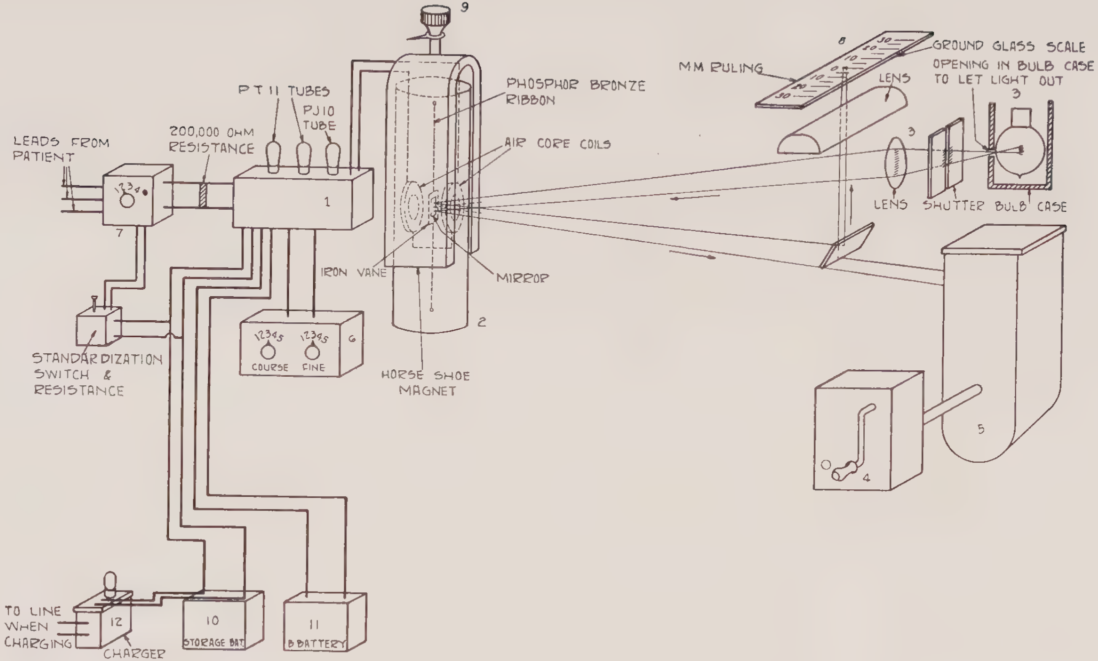

Fig. 60. Diagram of the Victor electrocardiograph.

can be used as long as their voltage output is 120 or higher and they will last from six to nine months, if the equipment is used one-half hour each day.

The galvanometer consists of two coils of wire wound on small bobbins with an air core, a phosphor bronze ribbon, iron vane, and mirror. As can be observed in the schematic diagram (Fig. 60) the air core coils are facing each other and slightly spaced. The phosphor bronze ribbon is suspended with a definite tension between the two coils. Fastened on this ribbon is the iron vane and attached to the vane is the small mirror.

The electrical variations of the heart, after being amplified, pass through the two air core coils and set up a varying field which acts upon the iron vane, causing it to turn one way or the other, depending upon the flow of current through the coils. Comparing the amount of twisting or turning of the iron vane to that of a complete circle, less than one-quarter of the diameter is used.

The coils and phosphor bronze ribbon are mounted in one assembly and immersed in oil. Each galvanometer is tested separately in a special device to determine the damping, and the viscosity of the oil is corrected in each galvanometer until correct damping is obtained. The entire galvanometer is sealed and does not require any attention. It should remain fixed in its adjustment indefinitely.

The spring motor is especially constructed for this apparatus. The speed is controlled very easily by an adjustment which controls a governor. This motor is so constructed that it will maintain a constant speed after winding for 25 feet of film. The speed of the film can be checked at any time the operator may care to do so by simply determining the time it takes for the film indicator to travel a certain number of feet (12 seconds per foot is the standard speed for electrocardiographic records).

The camera is so arranged as to be removable from the equipment and will hold 50 feet of film or bromide paper at one loading. The film or paper that has been exposed can be removed at any time without disturbing the unexposed portion. An indicator on the panel of the apparatus case informs the operator of the amount of film that is used.

The optical and lighting system consists of a concentrated filament bulb mounted in a case. The light is allowed to pass out of the case through a small oblong hole. A simple shutter is used to determine the width of the beam. It is possible for the operator to have a beam width of any dimension that he may desire up to 3 mm. The standard width is 1½ mm. A simple lens condenses the light upon the mirror from which it is reflected to the camera lens.

There is another mirror which intercepts a portion of the beam reflected by the galvanometer, deflecting it upward through a lens to the ground glass sensitivity scale. This permits the operator to observe at any time the action of the heart which is reproduced by the movement of the spot of light on the sensitivity scale.

No adjustments of the optical system are necessary excepting that of the bulb, when it is replaced. Due to the fact that the filaments in these bulbs do not all occupy exactly the same position, it is necessary, when replacing a bulb, to adjust it vertically and horizontally. This is accomplished by two screws on the apparatus case panel. Once set, further adjustment is not necessary. The object in view when setting the vertical and horizontal adjustment is to obtain the brightest light beam possible on the camera lens and ground glass screen.

A beam adjustment is provided to enable the operator to have accurate control of the position of the galvanometer mirror. This control consists of a permanent horseshoe magnet, mounted on the outside of the galvanometer case and so arranged, by gearing, that the position can be changed at will by turning the beam control knob. The magnetic field set up by this permanent magnet controls the position of the iron vane.

No adjustments or precautions in regard to body resistance are necessary with this equipment. No attention need be paid to body resistance. This is brought about by the fact that the equipment has inserted in the amplifying and patient circuit a resistance of 200,000 ohms. The addition of the patient and his resistance, which may vary from 1000 to 5000 ohms, cannot change this total to an appreciable extent.

Compensation for skin current or neutralization is not necessary. Skin current cannot damage the equipment and therefore it is not necessary to use protecting resistances. Should the light spot be deflected out of sight by the skin current when the patient is in the circuit, it is only necessary by manipulation of the beam control to bring the light back to center position.

There is one feature in the present model which will at times be a handicap in the clinical use of the instrument. This is the relative narrowness of the recording film or paper. Its width is only 45 mm. and occasionally one meets with patients who give a total amplitude of deflection which is so large that unless the zero level remains quite constant the deflections will shoot off the recording film. This difficulty is best met by manipulating the beam adjustment so that first the bottom of the deflection and then the top is brought within the central 2 cm. of the record. It is better to do this than to reduce the sensitivity of the galvanometer for this procedure will change the appearance of the electrocardiogram though of course not the proportionate size of the various excursions.

The records of this apparatus have no time marking. It would perhaps be more reassuring if there were something to show that the film is maintaining a constant speed, but in practical operation this seems to be unnecessary as the motor mechanism for driving the film does not change its speed. When this is set at 25 mm. per second it remains constant indefinitely as long as care is used to keep the spring wound up.

The following brief description will bring out the method of operating the equipment:

1. Attach electrodes to the patient as usual, except that the special precautions to insure low skin resistance are not necessary. The electrodes are labeled L A, R A, and L L, for left arm, right arm, and left leg.

2. Lead selector switch is to be placed on Contact 4. This

automatically disconnects the patient from the apparatus.

3. Push down main on-and-off switch. This turns on the entire apparatus and makes it ready for taking the heart record.

4. Allow 30 seconds to pass for the equipment to stabilize. This is necessary to give time for the two condensers, used in the amplifier, to charge. When the apparatus is stable, the light spot on the sensitivity scale will be stationary.

5. Press down standardization switch and it will be noticed that light spot will deflect on sensitivity scale. If the deflection is not 1 cm., adjust the two sensitivity controls which regulate the amplification until the deflection is 1 cm. Once set, the standardization will not change from lead to lead or from patient to patient. It will remain in standardization just as long as the storage battery is kept up to full charge.

6. Open the camera by turning the knob controlling the camera shutter.

7. Start the spring motor by turning the knob for that purpose. This causes the film to move. The speed is 25 mm. per second.

8. Press standardization switch once. This makes a record of the standardization on the film.

9. Stop the spring motor.

10. Turn lead selector to Lead "1." If the light spot on sensitivity scale goes off scale, bring it back by adjustment of beam control. The heart action may now be observed.

11. Start spring motor and allow as much film as necessary to be exposed. The amount of film exposed will be shown by the indicator.

12. Stop spring motor. Turn lead selector to Lead "2." Duplicate action as with Lead "1."

13. Turn lead selector to Lead "3" and duplicate action.

14. Close camera shutter.

15. Shut off main switch.

16. Remove camera and while in dark room remove film for development.

The clinical operation of this instrument is remarkably simple and free from technical difficulties, owing to the fact that there is no trouble from the patient's resistance and that compensation and standardization need not be performed with each lead. Careless operation of the instrument does not lead to any difficulty comparable to the breaking of a string in the Einthoven electrocardiograph.

APPENDIX I

Einthoven's tables for the determination of the direction within the heart of any current recorded by the three standard leads. These directions refer only to the frontal plane, so that currents which lie in other planes are only represented as they affect the frontal plane.

One must first make certain that the deflections measured in the three leads represent the same time instant of the electrocardiogram, for otherwise the deflections will not be due to the same current within the heart. This can often be approximated by an inspection of the record, measuring off with a magnifying glass the distance from the beginning or end of some sharp deflection such as Q, R or S. If we are dealing with deflections which represent the same current within the heart they will be found to fulfil very closely the formula:

Value Lead 1 + value Lead 3 = value Lead 2.

If they do not approximate this formula they do not arise from the same current within the heart.

The direction of any current within the heart is expressed in relation to the horizontal; the 180° below the horizontal being considered positive (+) and the 180° above the horizontal, negative (−). The direction 0° is horizontally to the patient's left and ± 180° horizontally to the patient's right (Fig. 10). Table III is the most satisfactory for ordinary use but Table II shows rather graphically the reciprocal variation of the lead values with changing direction of the vectors.

TABLE II

Angle α	Registered potentials reduced to a value of ± 10 for the largest wave			Manifest potential Em
	Lead 1	Lead 2	Lead 3	
− 80°	1.8	− 8.2	−10	10.7
− 70°	3.5	− 6.5	−10	10.2
− 60°	5.0	− 5.0	−10	10.0
− 50°	6.5	− 3.5	−10	10.2
− 40°	8.2	− 1.8	−10	10.7
− 30°	10.0	0.0	−10	11.5
− 20°	10.0	1.8	− 8.2	10.7
− 10°	10.0	3.5	− 6.5	10.2
0°	10.0	5.0	− 5.0	10.0
10°	10.0	6.5	− 3.5	10.2
20°	10.0	8.2	− 1.8	10.7
30°	10.0	10.0	0	11.5
40°	8.2	10.0	1.8	10.7
50°	6.5	10.0	3.5	10.2
60°	5.0	10.0	5.0	10.0
70°	3.5	10.0	6.5	10.2
80°	1.8	10.0	8.2	10.7
90°	0	10.0	10.0	11.5
100°	− 1.8	8.2	10.0	10.7
110°	− 3.5	6.5	10.0	10.2
120°	− 5.0	5.0	10.0	10.0
130°	− 6.5	3.5	10.0	10.2
140°	− 8.2	1.8	10.0	10.7
150°	−10.0	0	10.0	11.5
160°	−10.0	− 1.8	8.2	10.7
170°	−10.0	− 3.5	6.5	10.2
±180°	−10.0	− 5.0	5.0	10.0
−170°	−10.0	− 6.5	3.5	10.2
−160°	−10.0	− 8.2	1.8	10.7
−150°	−10.0	−10.0	0	11.5
−140°	− 8.2	−10.0	− 1.8	10.7
−130°	− 6.5	−10.0	− 3.5	10.2
−120°	− 5.0	−10.0	− 5.0	10.0
−110°	− 3.5	−10.0	− 6.5	10.2
−100°	− 1.8	−10.0	− 8.2	10.7
− 90°	0	−10.0	−10.0	11.5

SUPPLEMENTARY TABLE

	Degrees to be added to the angle when second column is read downwards	Value of the smallest lead when the largest is reduced to ± 10	Degrees to be added to the angle when second column is read upwards	Manifest potential
Section 1......	2°	±0.4	8°	11.3
	4°	±0.8	6°	11.1
	6°	±1.2	4°	11
	8°	±1.5	2°	10.8
	0	±1.8	0	10.7
Section 2......	2°	±2.2	8°	10.5
	4°	±2.5	6°	10.4
	6°	±2.9	4°	10.3
	8°	±3.2	2°	10.2
	0	±3.5	0	10.2
Section 3......	2°	±3.8	8°	10.1
	4°	±4.1	6°	10.1
	6°	±4.4	4°	10
	8°	±4.7	2°	10
	0	±5	0	10

To use Table II, the recorded potential is determined by measuring the height of the wave at the same time instant in each lead; the value of the smallest lead is made proportional to a value of 10 for the largest lead by substituting them in the formula:

Value largest lead : 10 :: value smallest lead : x

Thus if Lead 1 $= +3.2$, Lead 2 $= +12.5$, Lead 3 $= +9.3$, then the formula would be:

$$12.5 : 10 :: 3.2 : x \left(\frac{3.2 \times 10}{12.5} = x\right) \text{ therefore } x = 2.56$$

Considering the values Lead 1 $= 2.6$, Lead 2 $= 10$ we find in Table II that this relation of the lead values lies between 70° and 80°.

If it is desired to locate the angle with greater exactness than this, the supplementary table must be used. The appropriate section of this table is supposed to be inserted between the lines of Table II, and the angle taken from the first or third columns of this supplementary table to be added to the smaller of the two obtained from the main table.

Since the value of Lead 1 is + 2.6, Section 2 of the supplementary table is to be used, for the value + 2.6 lies within its limits. In Table II the value of Lead 1 at 70° is + 3.5 and at 80°, + 1.8, so the second column of the supplementary table must be read from + 3.5 to + 1.8, i.e., upward. The figure nearest to + 2.6 in this center column is + 2.5 and the angle opposite this in the third column is 6°. This angle is added to the basic 70° making 76° which is the exact angle to produce a vector with the designated proportional lead values.

Should we wish to determine the manifest potential (Em) that is, the hypothetical potential which would give rise to such lead excursions, we take the figure for manifest potential which lies in the supplementary table opposite the angle obtained from this table. In this case the figure is 10.4. This is the manifest potential for a maximum excursion of 10 mm., so that for the lead values with which we started, this figure must be changed according to the formula:

$$10.4 : 10 :: x : 12.5 \left(\frac{10.4 \times 12.5}{10} = x \right) \text{ therefore } x = 12.95$$

This is the potential which would give the lead excursions we have measured if its direction were 76° and there were no short-circuiting within the body.

I feel it necessary to state again here that this manifest value does not actually exist. Within the heart there are at any one instant many potentials in many different directions. The sum of all these is a certain potential having a certain direction, and this potential, reduced by short-circuiting within the body, gives rise to the excursions in the leads. The manifest potential has a value which is due to the short-circuiting of the summated heart potentials at a given instant. If there were no short-circuiting the current within the heart would be the manifest potential.

To use Table III to determine the angle of any potential difference within the heart from its registered potential in the leads (e1, e2 and e3) the smallest of these must be made proportional to a value of 10 for the largest. (Multiply the smallest value by 10 and divide by the largest

TABLE III—THE ANGLE ALPHA

Smallest value of ± e	Largest lead	$e_1 = +10$		$e_1 = -10$		$e_2 = +10$		$e_2 = -10$		$e_3 = +10$		$e_3 = -10$	
	Smallest lead	2	3	2	3	1	3	1	3	1	2	1	2
0.00	E 10 1.15	−30°	30°	150°	−150°	90°	30°	−90°	−150°	90°	150°	−90°	−30°
0.20	1.14	−29°	29°	151°	−151°	89°	31°	−91°	−149°	91°	149°	−89°	−31°
0.40	1.13	−28°	28°	152°	−152°	88°	32°	−92°	−148°	92°	148°	−88°	−32°
0.59	1.12	−27°	27°	153°	−153°	87°	33°	−93°	−147°	93°	147°	−87°	−33°
0.78	1.11	−26°	26°	154°	−154°	86°	34°	−94°	−146°	94°	146°	−86°	−34°
0.96	1.10	−25°	25°	155°	−155°	85°	35°	−95°	−145°	95°	145°	−85°	−35°
1.14	1.09	−24°	24°	156°	−156°	84°	36°	−96°	−144°	96°	144°	−84°	−36°
1.32	1.09	−23°	23°	157°	−157°	83°	37°	−97°	−143°	97°	143°	−83°	−37°
1.50	1.08	−22°	22°	158°	−158°	82°	38°	−98°	−142°	98°	142°	−82°	−38°
1.68	1.07	−21°	21°	159°	−159°	81°	39°	−99°	−141°	99°	141°	−81°	−39°
1.85	1.06	−20°	20°	160°	−160°	80°	40°	−100°	−140°	100°	140°	−80°	−40°
2.02	1.06	−19°	19°	161°	−161°	79°	41°	−101°	−139°	101°	139°	−79°	−41°
2.19	1.05	−18°	18°	162°	−162°	78°	42°	−102°	−138°	102°	138°	−78°	−42°
2.35	1.05	−17°	17°	163°	−163°	77°	43°	−103°	−137°	103°	137°	−77°	−43°
2.52	1.04	−16°	16°	164°	−164°	76°	44°	−104°	−136°	104°	136°	−76°	−44°
2.68	1.04	−15°	15°	165°	−165°	75°	45°	−105°	−135°	105°	135°	−75°	−45°
2.84	1.03	−14°	14°	166°	−166°	74°	46°	−106°	−134°	106°	134°	−74°	−46°
3.00	1.03	−13°	13°	167°	167°	73°	47°	−107°	−133°	107°	133°	−73°	−47°
3.16	1.02	−12°	12°	168°	−168°	72°	48°	−108°	−132°	108°	132°	−72°	−48°
3.32	1.02	−11°	11°	169°	−169°	71°	49°	−109°	−131°	109°	131°	−71°	−49°
3.47	1.02	−10°	10°	170°	−170°	70°	50°	−110°	−130°	110°	130°	−70°	−50°
3.63	1.01	−9°	9°	171°	−171°	69°	51°	−111°	−129°	111°	129°	−69°	−51°
3.78	1.01	−8°	8°	172°	−172°	68°	52°	−112°	−128°	112°	128°	−68°	−52°
3.94	1.01	−7°	7°	173°	−173°	67°	53°	−113°	−127°	113°	127°	−67°	−53°
4.09	1.01	−6°	6°	174°	−174°	66°	54°	−114°	−126°	114°	126°	−66°	−54°
4.24	1.00	−5°	5°	175°	−175°	65°	55°	−115°	−125°	115°	125°	−65°	−55°
4.39	1.00	−4°	4°	176°	−176°	64°	56°	−116°	−124°	116°	124°	−64°	−56°
4.55	1.00	−3°	3°	177°	−177°	63°	57°	−117°	−123°	117°	123°	−63°	−57°
4.70	1.00	−2°	2°	178°	−178°	62°	58°	−118°	−122°	118°	122°	−62°	−58°
4.85	1.00	−1°	1°	179°	−179°	61°	59°	−119°	−121°	119°	121°	−61°	−59°
5.00	1.00	0°	0°	180°	−180°	60°	60°	−120°	−120°	120°	120°	−60°	−60°

value.) Compare the figure thus obtained with those in the first column of the table and the angle will be on the *horizontal* line whose number is closest to the figure. The angle will be found at the intersection of this line with the *vertical* column determined by a combination of two leads, those with the smallest and the largest recorded values, i.e., whether the largest value was + or − in Lead 1, Lead 2, or Lead 3 ($e1 = 10$, $e1 = -10$, $e2 = 10$, $e2 = -10$, etc.) and by the lead in which the smallest value occurred.

For example, if the values in the leads were $e1 = 16$, $e2 = 10$, $e3 = -6$, then by using the above formula we obtain $\left(\frac{6 \times 10}{16}\right) = 3.75$. The angle is therefore found on the line with 3.78 in the first column, as this is the figure closest to 3.75. Since the largest value was a + value in Lead 1, the vertical column will be under $e1 = +10$ and in the particular column under 3, since the smallest value was in Lead 3. The angle in this case is 8°. Again, if the values are 8, −4 and −12, the horizontal line will be opposite 3.32 $\left(\frac{4 \times 10}{12} = 3.33\right)$ and the vertical column will be that of Lead 2 (the lead of smallest value) under $e3 = -10$ (the lead of largest value). The angle is −49° in this case.

This table is much quicker to use than Table I, for it does away with the need of a supplementary table.

To obtain the manifest potential from Table III is very simple, for the figure in the column under E10 which lies on the same line as the angle we obtained is to be used just as was the similar figure obtained from the supplementary table of Table II. The largest recorded value in the leads is placed in the formula $E10 : 10 :: X :$ largest value. For the first example above, the manifest potential would be 16.64 and for the second example it would be 12.24.

APPENDIX II

METHOD OF FILING AND INDEXING ELECTROCARDIOGRAPHIC RECORDS

Owing to their size and character, the preservation and filing of electrocardiographic records presents various difficulties. It seems worth while for this reason to give in detail the method which has gradually been evolved at the New York Hospital and which in its present form gives considerable satisfaction. It enables one to find promptly the record of any patient or to pick out with minimum effort a series of electrocardiographic records showing any peculiarity tabulated. The three strips, Lead 1, Lead 2 and Lead 3, are placed in order face downward and fastened together with adhesive linen tape. They are then placed in ordinary cardboard folders so that Lead 1 lies at the top. The name of the patient is written on the upper edge of the folder, the number of the record and the date on the lower flap. Subsequent records of the same patient may be placed in this folder, so that all records of the same patient are readily found together. Folders are filed alphabetically according to names of patients.

The legal size cardboard folder, 9¾ by 15 inches, is used because the great length of this container enables us to file long strips of the record without folding. It has been found that when the record has been folded over it eventually cracks and tears at the point of folding. Another reason for using the long folder is that it gives space for the filing signals which are later to be placed along its edge.

A book is kept in which, opposite the serial number of the record, is written the name of the patient, the age, the clinical diagnosis, the blood pressure and the amount of digitalis which had been given during the two weeks previous to taking the record. Quinidine or morphin medication during the twenty-four hours previous to the record is also noted. These are the facts which the physician who interprets the record must have in mind, in order to make his report as helpful as possible to the clinician. Without them the report will not reveal its full usefulness.

The accompanying diagnosis table is used in diagnosing the records. It will be found that by combining the numbers of these diagnoses with a very few words it will be possible to write a satisfactory report of any electrocardiogram. These diagnosis numbers are written by the physician in the book under the patient's name and serial number. About 4 inches of each lead is torn off and the serial number and the number of the lead marked on the back. In order that the secretary may know which way to mount the pieces of the record it has been found satisfactory to turn the strip over as one would turn a check for endorsement and write the number and lead on the back of the left-hand end of the strip. The book is now turned over to the secretary who mounts the pieces of each lead and types the report of the record somewhat as follows. A report in the book reads thus:

4 rate 110 – 8 a1 (P – R = .22) – 14 marked – 12 a bn 1 bs 2 and 3 d – 16 a – 22.

It would be written as follows:

Simple tachycardia rate 110 per minute
Partial auriculoventricular heart-block with prolonged conduction time (P – R = .22 second)
Marked left axis deviation of Q R S
Q R S is abnormally wide and shows notching in Lead 1 and slurring in Leads 2 and 3
The Q R S voltage is unusually small
The T wave is turned downward in Lead 1
These abnormalities indicate disease of the ventricular muscle.

Again:

7 (rate 95) – occas 3c – 13(12 bs 3 = 28) – 16 b – 21 – later record requested.

This would be written as follows:

Auricular fibrillation, heart rate 95 per minute
Occasional ventricular premature beats
Right axis deviation of Q R S

Q R S group is slurred in Lead 3, but this is of no clinical significance

T wave turned downward in Lead 2; this T wave abnormality may be due to digitalis or to disease of the ventricular muscle

A later record is requested

This system saves a great deal of time in reporting the records, but its chief value lies in the ease with which it adapts itself to indexing any peculiarities which may be considered important. For this purpose colored signals are placed upon the upper edge of the folder, the position and color of the signal indicating the peculiarity. The Graffco vise signals which are of metal and clamp upon the edge of the folder have been found satisfactory, though cardboard strips may be used if pasted on. Signals colored red, green, pink, white, orange and blue are used and these colors are repeated in this order from left to right so that 48 of the signals may be set edge to edge across the top of the folder. In actual practice folders usually have but two or three signals and rarely as many as six. A color key is made upon a piece of card, the colors being painted in their proper order for 15 inches and the number of the peculiarity to be recorded being marked below its corresponding color. This card is used as a guide for placing the signals properly and also for picking out records with any desired peculiarity. The features indexed in this way at the New York Hospital are indicated in the diagnosis table by having the initial of one of the color signals in parentheses after it. These are the features which seem especially important for a cross index, but obviously any list of features could be used in this way.

Diagnosis Table

1. Normal sinus rhythm
2a. Sinus arrhythmia (r)
 b. Sinoauricular block (g)
 c. Simple bradycardia (p)
 d. Ventricular escape (w)
3. Premature beats
    a. Auricular (o)
    b. Nodal (b)
    c. Ventricular (r)
    d. Unusual premature beats (g)
4. Simple tachycardia (p)

5. Paroxysmal tachycardia
   - a. Auricular (W)
   - b. Nodal (O)
   - c. Ventricular (B)
6. Auricular flutter
   - a. Paroxysmal (R)
   - b. Chronic (G)
7. Auricular fibrillation
   - a. Paroxysmal (P)
   - b. Chronic (W)
8. Auriculoventricular heart-block
   - a1. Partial block, prolonged conduction }
   - 2. Partial block, dropped beats } (O)
   - b. Complete block (B)
   - c. This prolonged conduction may be due to digitalis or disease of the bundle
9. Normal A-V conduction time
10. Rhythm undiagnosed (R)
11. P Wave
    - a. Abnormally wide (G)
    - b. Notched (P)
    - c. Voltage unusually large (W)
    - d. Voltage unusually small (O)
    - e. Unusual peculiarity (B)
    - ee. Inversion of
12. Q R S Group
    - a. Abnormally wide (R)
    - bn. Notched }
    - bs. Slurred } (G)
    - bn & s. Notched & slurred }
    - c. Voltage unusually large (P)
    - d. Voltage unusually small (W)
    - e. Unusual peculiarity of (O)
13. Right axis deviation of Q R S (B)
14. Left axis deviation of Q R S (R)
15. T wave
    - a. Prolonged (G)
    - b. Notched (P)
    - c. Voltage unusually large (W)
    - d. Voltage unusually small (O)
    - e. Unusual peculiarity }
    - ee. Unusual voltage of S-T interval } (B)
    - f. T wave shows effect of digitalis (R)
    - g. Diphasic in Lead 1 (G)
    - h. Diphasic in Lead 2 (P)
    - i. Diphasic in Leads 1 and 2 (W)
16. T wave turned downward
    - a. Lead 1 (O)
    - b. Lead 2 (B)
    - c. Leads 1 and 2 (R)
    - d. Coronary T wave (G)

17. Bundle-branch block (P)
18. Intraventricular block (W)
19. Neither right nor left axis deviation
20. No abnormality of ventricular waves to suggest myocardial disease
21. This T wave abnormality may be due to digitalis or to disease of the ventricular muscle
    a. Probably the former
    b. Probably the latter
22. These abnormalities indicate disease of the ventricular muscle
23. Indicating disease of ventricular muscle
24. Indicating poor functional condition of the muscle
25. The waves are deformed by the presence of high skin resistance
26. The interpretation of this record is unsatisfactory due to digitalis patient has received
27. Of doubtful significance
28. Of no clinical significance

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